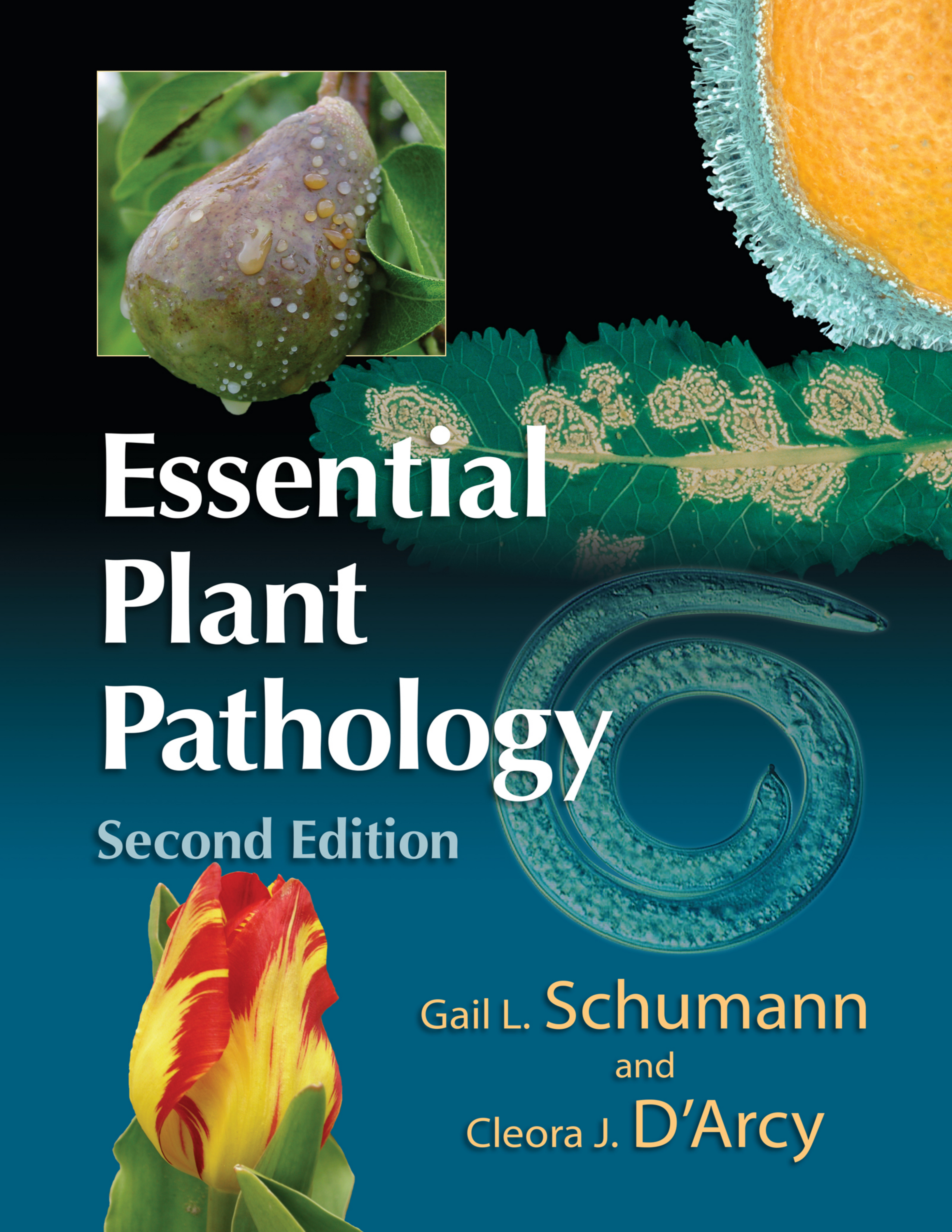

Essential Plant Pathology

Second Edition

Gail L. Schumann
and
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Second Edition

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Cover: Upper left, fire blight, caused by *Erwinia amylovora*, on pear; courtesy M. Bulatovic-Danilovich, reprinted from the cover of *Plant Disease*, Vol. 87, No. 5.
Upper right, blue whisker mold, caused by *Penicillium ulaiense*, on orange; courtesy G. J. Holmes, reprinted from *Plant Disease*, Vol. 77, No. 5.
Center, white rust, caused by *Albugo candida*, on brassica; courtesy P. A. Williams, copyright-free.
Lower right, spiral nematode, *Heliocotylenchus* sp.; used by permission of Mactode Publications, photo by Urlich Zunke.
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► **To Mike and Steve, with love**

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Preface

Why Study Plant Pathology?

Plant pathology is the study of plant diseases. We could not survive on earth without plants, so their health is important to us. Photosynthesis is the source of the free oxygen necessary for aerobic respiration. Long ago, free oxygen became a component of the early atmosphere, leading to the creation of the ozone layer, which helps to protect us from ultraviolet radiation and makes life on land possible. Photosynthesis also is the source of fixed carbon, from which all organic molecules (including the components of our bodies!) are then produced. Directly or indirectly, plants provide all of the food on which the human population relies. In addition, plants provide feed for animals, shelter, fiber for clothing and paper, fuel, and ornamental flowers and landscape plants for our enjoyment.

Plant diseases reduce yields of food and cash crops, mar the beauty of ornamental plants, and reduce our ability to shelter and feed ourselves. In some cases, pathogens even produce toxic compounds that poison our food. It is impossible to separate plant diseases from the history and culture of humans. Plant diseases limit where crops can be grown and determine what foods are available to us. A single disease, late blight of potato, triggered the Irish potato famine of the 1840s and forever changed the history of Ireland and of North America, where many starving Irish peasants took refuge. Dutch elm disease has killed more than 5 million elm trees on the formerly tree-lined streets of cities in the United States and Europe. Citrus canker and soybean rust currently threaten multibillion dollar crops in the United States and around the world. Each year, local, state, and federal governments spend funds on quarantines and plant inspection services to control the movement of dangerous pathogens that threaten our food supply and the livelihoods of growers. The concern that bioterrorism may threaten genetically uniform crops, as well as farm animals and human populations, has made us more aware of the potential for damage by introduced pathogens. Billions of dollars are spent each year on the management of plant diseases, and yet it is estimated that plant diseases cause a 20% yield loss in food and cash crops. Of course, that does not mean that all yields are reduced by that amount. Some crops remain healthy, but others may be nearly destroyed, potentially causing starvation for large populations of people or loss of livelihood for farmers.

Aside from the practical aspects of keeping plants healthy, plant pathology is an amazing biological science that focuses on the interactions among host plants, pathogens, and the vectors that transmit the pathogens in various environments. Studies of the physiology and genetics of these interactions lead us to new discoveries in molecular biology and their applications in biotechnology. Knowledge of the ecological interactions of hosts and pathogens will help us understand our natural ecosystems, global climate change, and maintenance of biological diversity on our planet. All aspects of biology are encompassed in the study of plant pathology. This textbook and its accompanying online resources are designed to introduce students to the fascinating study of plant diseases.

How to Use This Textbook and Online Resources

The second edition of this textbook was reviewed by subject matter specialists and plant pathology instructors to improve its accuracy and update the information. Many new APS Education Center publications and web links have been added to the online resources. Links have been added to the online resources that go directly to the index of Plant Disease Profiles and to the Illustrated Glossary. The Illustrated Glossary has been updated and revised. A printed version of the glossary has been added to the textbook, but the online version is recommended because it includes drawings and images. The identification exercises in Chapters 2 and 8 have been expanded and revised. Additional tropical crops and their diseases have been added to Appendix 2 for use by students doing Internet research and other exercises.

Hints for Students

Textbook Resources. Summaries are provided in bold type at the ends of sections. The purpose of these summaries is to help you to determine the important points of the preceding section and to find information more easily when you review the text of each section. Within sections of text are images, illustrations, content summary boxes, and Disease Classics. Disease Classics are short summaries of important diseases that are typically studied in introductory courses. Most of the Disease Classics also exist as Plant Disease Profiles in the APS Education Center, where you will find full disease cycles, color photographs, and more detailed information. Appendix 1 is a list of the Disease Classics. Some boxes within the text have the heading “Did you know?” These are interesting cultural and historical facts about plant diseases. At the end of each chapter in the textbook, you will find recommended resources, study questions, Words to Know, online exercises, and Internet research exercises.

Additional Resources. These include print references, titles of APS Education Center publications, and direct links to additional online references and websites that require Internet access. The textbook provides the basic information related to the subjects in each chapter, but most students want to learn more about certain topics or about specific diseases that attack plants of interest to them. These resources provide color photographs and the additional information to help students do this.

Study Questions. Standard review questions and challenge questions are provided in the textbook. You should be able to answer the standard review questions when you complete the materials for each chapter. They will help you prepare for tests. To answer the challenge questions will require some research outside the textbook. Challenge questions are designed to help you explore beyond the basic information in the textbook.

Words to Know. Important new vocabulary words are printed in bold in the textbook. One of the hardest parts of beginning any new field of study is to learn its vocabulary. We are trying to make this easier by indicating which words are important and by providing direct links from the online Words to Know to the definitions in the Illustrated Glossary. The Words to Know section at the end of every chapter also links to the online glossary. Most of the definitions have illustrations or photographs that make the new words easier to understand and remember.

Online Exercises. These are designed primarily for identification practice. Included in these exercises are important symptoms, signs, vectors, and common diseases that beginning plant pathologists should know. Answers are provided. The exercises and images are provided in the online resources.

Internet Research. You may have an interest in a specific crop or plant type, and the Internet research exercises have been designed to help you begin to study diseases that affect that commodity at the very beginning of your plant pathology course. As a starting point, Appendix 2 provides a list of common diseases of some economically important plants. Direct links to recommended websites are found in the descriptions of the Internet research exercises.

Plant pathology is a rapidly changing and important field of biology. Although this textbook is designed to provide the "essentials," there is much more to learn. We encourage you to continue to read, consult online resources, and take additional courses for a comprehensive education in plant pathology.

Hints for Instructors

Plant pathology courses vary in length and topics covered. Some include a laboratory component, and others do not. This textbook is arranged to meet the needs of students in these varied courses. The materials are presented in various formats, and students are asked to complete a variety of exercises. These provide opportunities for students with different learning styles to master the material.

This book should be used along with the resources available in the APS Education Center. All APS Education Center publications are peer-reviewed. Although this textbook refers students to existing publications, students should be encouraged to explore the more recent publications. Direct web links are included for additional materials, so students can access them easily.

CONTENTS

This textbook begins with an introduction to plant diseases and covers some basic concepts and vocabulary, illustrated by a case study of apple scab. Chapters 2–6 cover the major pathogen groups. We have tried to write these to be independent chapters, so they can be taught in the order preferred by the instructor.

A special note for Chapter 2. The fungal terminology taught in introductory courses varies, often depending on whether the course includes a laboratory or not. The first section of Chapter 2 emphasizes the basic biology and terminology required to understand fungi as plant pathogens. More detailed biology and terminology are in the second section of Chapter 2 for instructors who wish to include this material.

Chapter 7 describes abiotic problems. Chapter 8 describes plant diseases organized by symptoms because many students in introductory courses think about plant diseases based on their impact on the plant rather than according to the type of pathogen that causes the disease.

Chapter 9 helps students understand host-pathogen relationships: ecological, physiological, and genetic. The final two chapters are designed to help students understand plant disease epidemics, how human beings affect plant diseases and are affected by them, and the management of plant diseases.

INSTRUCTOR RESOURCES

Textbook Illustrations. Electronic versions of all textbook images, both photographs and diagrams, are provided in the online Instructor Resources for use in classroom presentations. For some illustrations, additional identification information has been added for instructor use.

Content Summary Boxes. These are all reproduced in the online Instructor Resources for classroom presentations.

Study Questions. Instructors may wish to recommend certain questions for exam preparation or class discussions. Challenge questions require more information than is provided in the textbook and may be appropriate for group assignments.

We hope you find these new instructional resources for the study of plant pathology exciting and useful. We welcome your comments and suggestions.

Acknowledgments

We have enjoyed teaching plant pathology courses over many years and have learned much from our students and their stimulating questions. Plant pathology instructors gave us advice and helpful comments both at the early stages of writing (E. J. Braun, E. L. Davis, J. Fletcher, A. B. Gould, K. N. Lambert, T. D. Murray, P. Vincelli, as well as some anonymous reviewers) and in editing the final versions of chapters (R. C. Gergerich, A. B. Gould, T. D. Murray, and M. Putnam.) For the second edition, we gratefully acknowledge M. L. Daughtrey, who reviewed the entire manuscript, and the following people who reviewed individual chapters: A. M. Alverez, R. M. Bostock, E. J. Braun, E. L. Davis, L. L. Domier, D. M. Eastburn, B. W. Falk, J. Fletcher, F. E. Gildow, A. B. Gould, D. C. Gross, R. Hammerschmidt, T. L. Niblack, D. L. Nickrent, A. C. Schilder, and P. Vincelli. Valuable suggestions were also provided by A. B. Baudoin, F. E. Brooks, Y. Cho, G. L. Coaker, G. S. Gilbert, D. I. Guest, and C. Hong. We also thank the numerous authors of instructional materials published in *The Plant Health Instructor* of the APS Education Center, which are listed as recommended materials in the textbook and in the online resources. Many other colleagues have contributed images and information; we especially acknowledge D. M. Eastburn, J. K. Pataky, and H. D. Thurston. Artists Vickie Brewster and Nancy Haver created the diagrams for this book. All contributors of figures and images are acknowledged

in a section at the end of the book. It is impossible to individually thank all of the colleagues who have made our professional lives so rich, but we hope that this textbook reflects our love of the science of plant pathology. We hope that it will aid many excellent plant pathology instructors as they inspire the next generation of plant pathologists. We also are thankful for the love and support of our families, who give balance and special meaning to our lives.

Gail L. Schumann
Cleora J. D'Arcy

CHAPTER 1

What Is Wrong with My Plant?

IN THIS CHAPTER:

- **How do we diagnose plant diseases?**
- **What is the plant disease triangle?**
- **How do we know what organism causes a disease?**
- **What are biotrophs and necrotrophs?**
- **What are disease cycles and how can we use them?**

CASE STUDY: APPLE SCAB

After a rainy spring, many of the apples in an orchard are scabby and deformed (Figure 1.1). Some have fallen to the ground, and the trees are nearly defoliated.

This is where we will begin to learn about plant diseases. What is wrong with these trees? And how can we prevent it from happening in the future?

The trees in this orchard have **apple scab**, one of the most important diseases of apples. This disease is caused by a fungus, *Venturia inaequalis*. Apple scab will be used as a case study in plant pathology, to illustrate concepts introduced in this chapter.

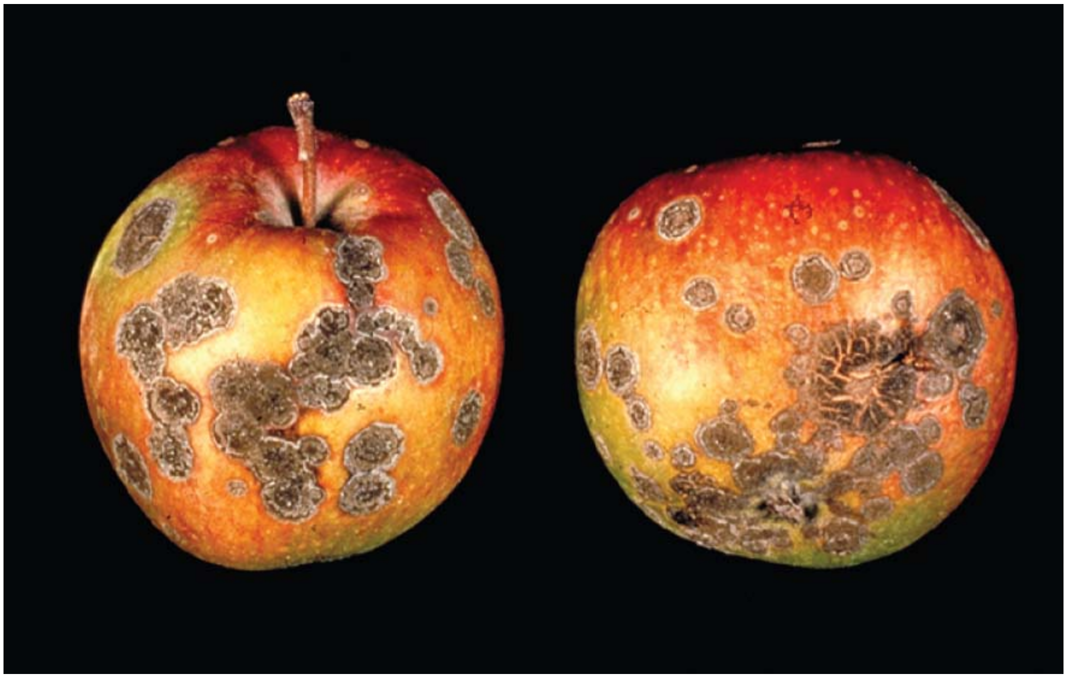


Figure 1.1. Apple scab symptoms on fruit.

Diseases of plants, like diseases of humans and other animals, are complex phenomena. A simple definition of **disease** is the abnormal functioning of an organism. One important characteristic of plant diseases is that they are injurious, causing harm to plants in some way. Disease also is progressive, which helps distinguish disease development over time from an instantaneous injury such as lightning striking a tree. Plant diseases are a challenge to people interested in maintaining and producing healthy plants. Plant health can be achieved only with an understanding of plant diseases. We need to know what causes them, how they spread, and how we can prevent them or at least minimize the damage they inflict.

► DID YOU KNOW?

Yields of the principal food and cash crops worldwide are reduced by almost 20% because of damage caused by plant pathogens.

Diagnosis is the first step in addressing the challenge of plant diseases. We are familiar with common symptoms and signs of animal and human diseases, but symptoms of plant diseases are very different from human symptoms. Physicians are able to question their patients and learn more about the symptoms and progression of their illnesses, but this is not possible with plants because they obviously can't answer our questions. For both human and plant diseases, symptoms alone are usually not sufficient for accurate diagnosis because the same symptoms can be caused by different **pathogens**, or disease-causing agents.

DISEASE CLASSIC

Apple Scab

Hosts apple, crabapple

Pathogen *Venturia inaequalis*, an *ascomycete* (a fungus)

Symptoms and Signs Olive green superficial lesions on leaves and flowers are the first symptoms. Later, scabs form on developing apples. When the disease is severe, defoliation may occur, and fruit may be cracked and deformed.

Disease Cycle The fungus overwinters in fallen apple leaves. In spring, sexual fruiting bodies (pseudothecia) produce ascospores (primary inoculum), which are forcibly discharged for dispersal through the air to newly expanding leaves. Conidia, the asexual spores (secondary inoculum), are produced on leaf lesions and dispersed by water to other leaves and developing fruit. Infections occur primarily in cool, wet weather but can continue throughout the season whenever an extended wet period occurs. Apple scab is a polycyclic disease.

Management Some resistant apple and crabapple cultivars are available. Fungicide application is the primary means of management in established orchards. Removal or destruction of fallen leaves, on which the pathogen overwinters and produces primary inoculum, can be effective for small numbers of trees. Research on how to make this method economically viable in orchards is under way.

Significance Apple scab is the most economically important disease of apple in many areas.

Plant Disease Profiles: Apple scab

Illustrated Glossary

Definitions and illustrations of common plant disease symptoms are presented in the Illustrated Glossary of Plant Pathology.

In this chapter, we will consider the **disease cycle** (the succession of events that occurs during the development of a disease) and the **disease triangle**. Plant pathologists apply these concepts in the diagnosis and management of plant diseases. This chapter introduces general concepts, and you will learn the details in the chapters that follow.

Plant diseases can seem very unfamiliar, even to people who are very familiar with plant culture, weeds, and insect pests. This is partly because many of the pathogens that cause plant diseases are microscopic. It is surprising, however, how much can be seen with the unaided eye or a hand lens after a little instruction. Although plant pathology novices may expect to learn how to cure plant diseases, most diseases can only be prevented. However, a better understanding of disease development can help minimize the problems caused by plant diseases.

How do we diagnose plant diseases?

Plant diseases have probably existed as long as plants have existed. Ancient records indicate that plant diseases have plagued humans since agriculture began. Blasts, blights, mildews, and rusts are mentioned in historical writings, including the Bible.

Most ancient people interpreted epidemics as punishment from their gods or accidents of fate. Even though some careful observers noted an increase of disease in low-lying areas where moisture accumulated or outbreaks of disease in the presence of microscopic organisms on infected plant parts, most people clung to the notion of **spontaneous generation** until the mid-1800s. Microscopes had been available since the 1600s, but people believed that the bacteria, fungi, and nematodes they observed in diseased tissues had spontaneously arisen and were the result of disease rather than its cause.

The science of plant pathology did not really begin until the mid-1840s, when devastating failures of potato crops caused hunger among peasants all over Europe and led to the Irish potato famine. The crops were destroyed by epidemics of late blight, a disease caused by a fungus-like organism, *Phytophthora infestans*. In 1861, Anton deBary published the results of the first controlled experiments proving that the fuzzy white growth (*P. infestans*) observed on the leaves of diseased potato plants (Figure 1.2) was the cause of the rot rather than the result of excessive rains, electricity from the new railroads, or various other factors proposed as explanations at the time. In the 1860s, experiments conducted by Louis Pasteur demonstrated that microorganisms are present in the air, refuting the concept of spontaneous generation. Further experiments led to the **germ theory** of disease, which states that specific microorganisms cause diseases. Until the causes of diseases were discovered, it was not possible to develop appropriate management strategies, and crop epidemics were problems without solutions. Disease diagnosis is the important first step in solving these problems.

► DID YOU KNOW?

Anton deBary (1831–1888), considered to be the "father of plant pathology," conducted some of the first studies of the life cycles of fungi that are important plant parasites. In 1861, he published the results of experiments that proved that *Phytophthora infestans* causes late blight of potato, essentially establishing the germ theory of disease in plants. This was well before Robert Koch's experiments with the animal disease anthrax that began in 1873.

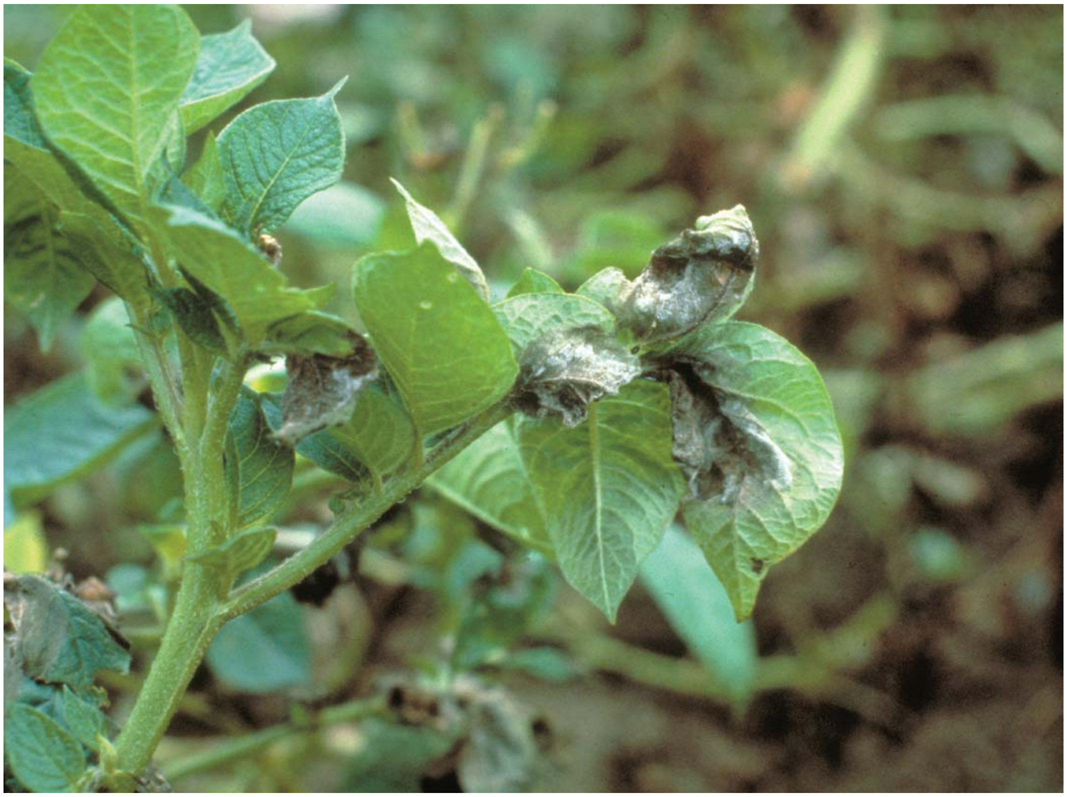


Figure 1.2. Potato plant with late blight.

DISEASE CLASSIC

Late Blight

Hosts potato, tomato

Pathogen *Phytophthora infestans*, an oomycete (a fungus-like organism)

Symptoms and Signs Water-soaked lesions develop rapidly into blight on leaves and stems, which become covered with white mildew. Infected tubers are reddish or purplish in the early stages but completely rot after invasion by secondary organisms, such as soft rot bacteria.

Disease Cycle In wet weather, sporangia (spore-bearing structures) of the pathogen produce zoospores (asexual spores), which can swim to infection sites and develop into mycelium. Sporangia can be carried by air or water to new plants or washed through the soil to infect tubers. Late blight is a polycyclic disease. When both mating types of the pathogen are present, thick-walled oospores (sexual spores) are produced. Oospores are the result of genetic recombination and serve as survival spores. When only one mating type is present, the pathogen survives as mycelium in tubers.

Management Late blight management strategies include deployment of genetic resistance, application of fungicides, disposal of diseased potatoes at harvesttime, and killing vines before harvest to prevent infection of tubers.

Significance Late blight is the disease that led to the Irish potato famine in the 1840s. Of a population of 8 million Irish, at least 1 million died and 1 million emigrated, primarily to Canada and the United States. For nearly 150 years, potato growers around the world managed a single mating type of the pathogen. In the 1980s, a second mating type was detected, as well as isolates that were resistant to the commonly used systemic fungicides. This led to major losses and new difficulties in managing the pathogen, which is now capable of rapid genetic change and survival as oospores.

Plant Disease Profiles: Late blight of potato and tomato

Accurate diagnosis requires consideration of the basic functions of the plant that are affected by disease. Plants, unlike animals, produce their own food by photosynthesis. Any pathogen that affects foliage will reduce the rate of photosynthesis and thus diminish the ability of the plant to produce food for itself. Nutrients produced by photosynthesis are distributed to all parts of the plant through the phloem, but some pathogens invade the phloem and interfere with its function. Plants continuously absorb water and minerals through their roots and transpire water through stomata in their leaves. Foliar pathogens, by killing epidermal cells or disrupting leaf tissues, can limit the ability of plants to control transpiration. Water and minerals are carried in the xylem, but some pathogens invade the xylem, interfering with the movement of water in the plant. Other pathogens disrupt root function. Most plants remain upright because they are supported by herbaceous or woody stems, but the phloem and vascular cambium in stems are soft, vulnerable tissues that can be invaded by canker pathogens. A group of specialized pathogens can cause wood decay when they metabolize lignin, the substance that gives wood its strength. Thus, plants can be diseased in many different ways.

All of the basic structures of plants are vulnerable to attack by pathogens. Disease symptoms reflect the basic functions of the plant that are affected.

What is the plant disease triangle?

The disease triangle (Figure 1.3) represents the three fundamental elements required for disease in plants:

- a susceptible plant
- a pathogen capable of causing disease
- a favorable environment

If any of these three elements is missing, no disease occurs.

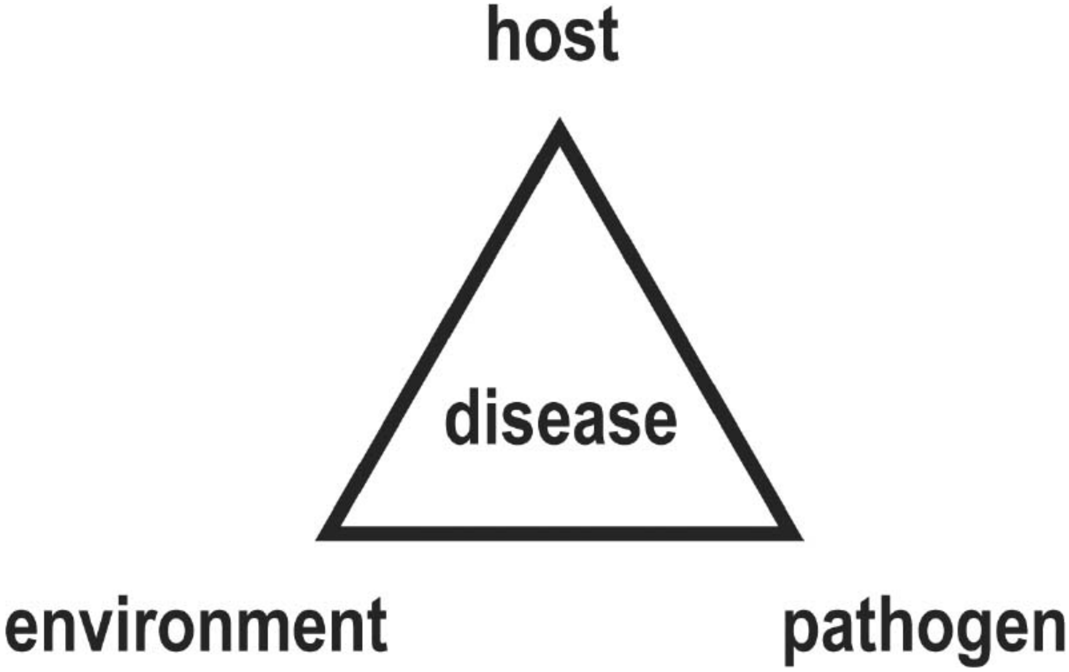


Figure 1.3. The disease triangle.

The Plant

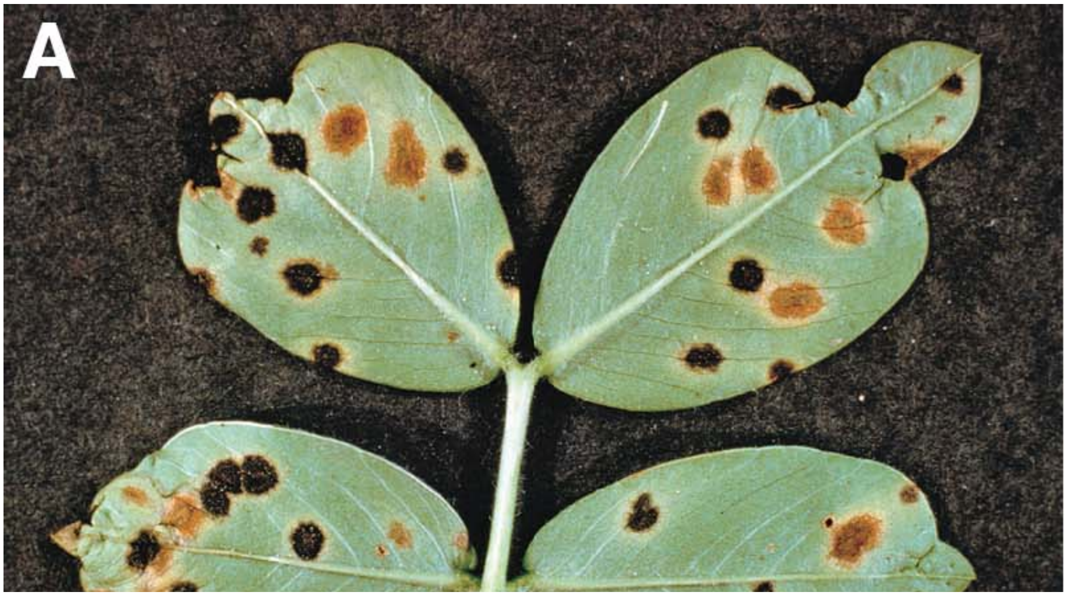
Susceptibility to disease can be affected by many factors, including the growth stage of the plant, its genetic predisposition, and stress. By careful observation of a diseased plant, one can begin to speculate about what basic functions of the plant are affected and, therefore, what kind of disease it may have.

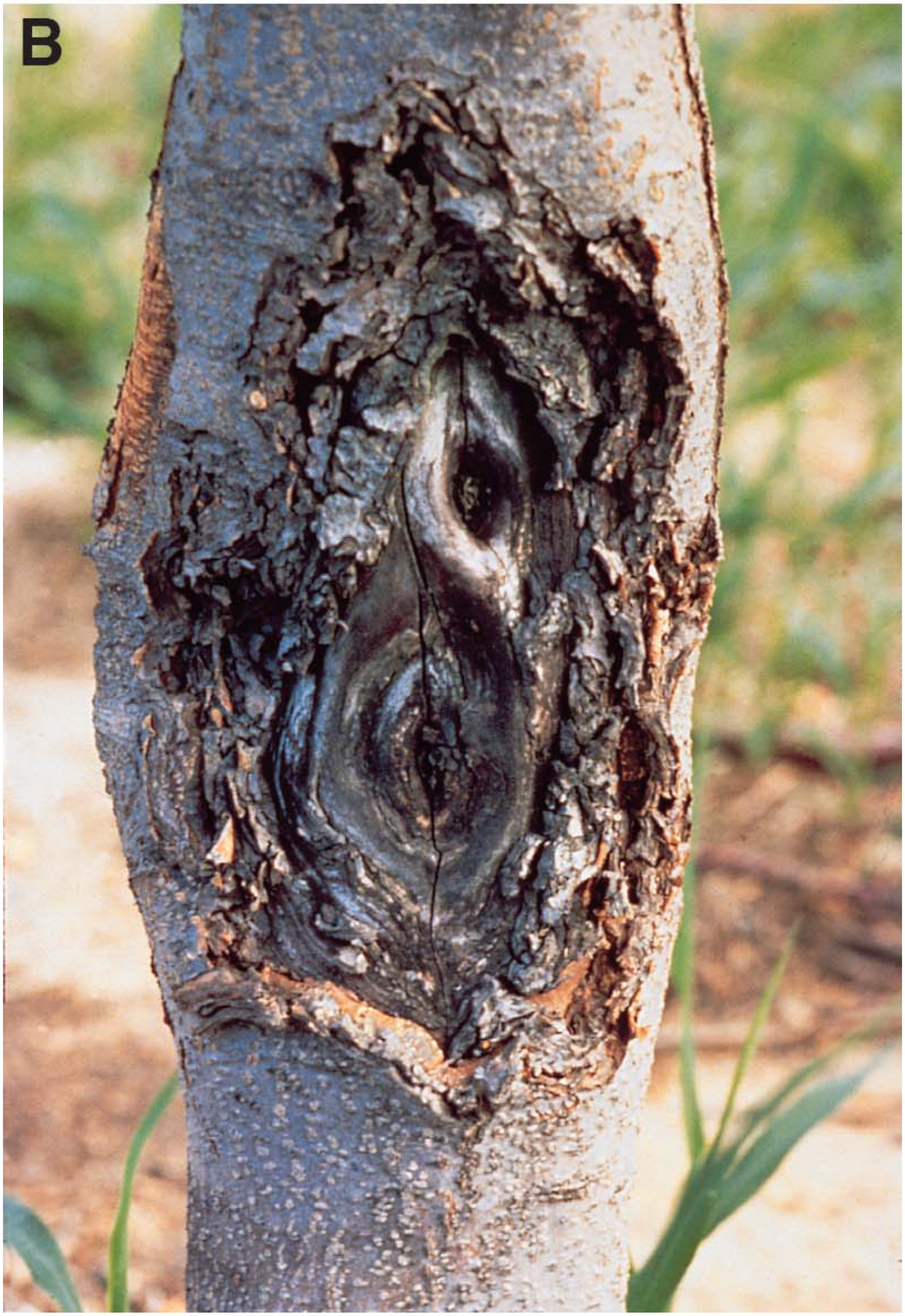
Symptoms are the expression of disease by a plant as a response to the activities of the pathogen. Symptoms may be localized, such as **leaf spots** (Figure 1.4A), or systemic, such as **stunting** that affects the entire plant. Some symptoms develop first on young and otherwise healthy tissues; others occur first on senescent tissues, such as aging flowers or lower leaves that are turning yellow.

CASE STUDY: APPLE SCAB

Symptoms

Apple scab affects only aboveground parts of the plant. Symptoms are most obvious on leaves, flowers, and fruit. Spots develop on leaves, and the leaves may become distorted and puckered. Lesions on leaves and fruit generally appear blistered and "scabby," with a distinct margin between healthy and diseased tissues. Severely infected fruit are distorted and cracked, and both leaves and fruit may prematurely fall from the tree. Disease may be observed on all susceptible plants in an area. Some apple and crabapple cultivars are resistant to the disease, and little or no apple scab develops in them.

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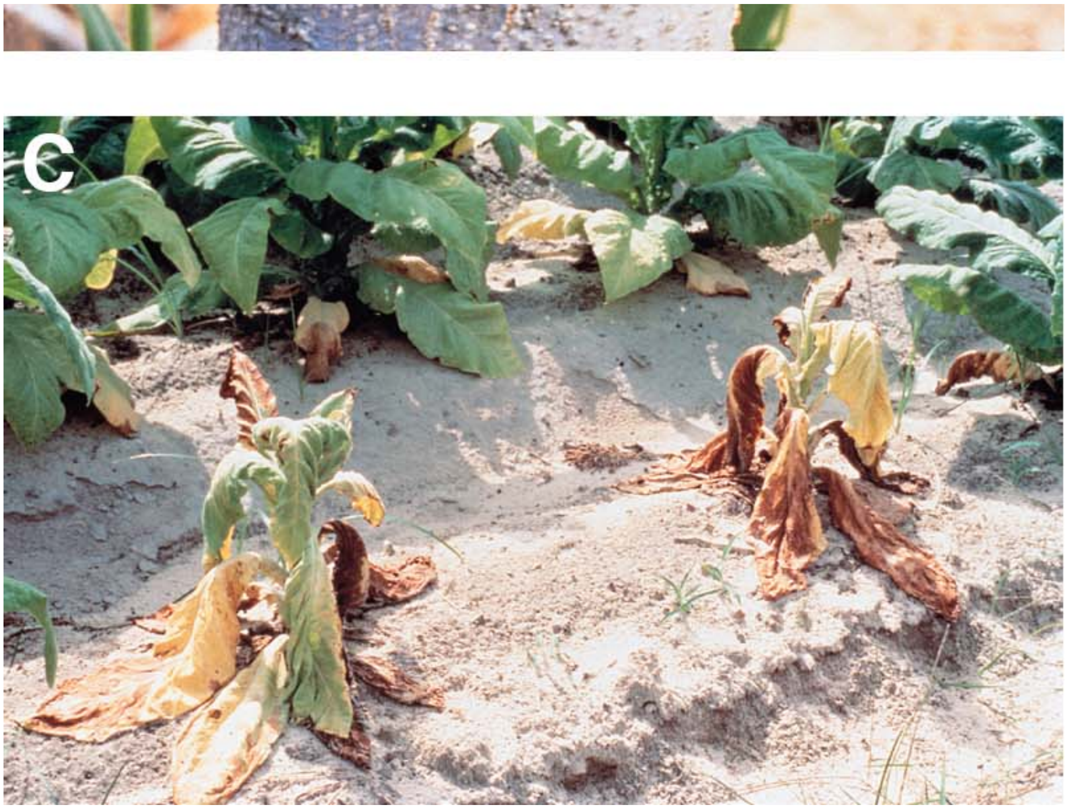


Figure 1.4. Common symptoms of plant diseases: A, leaf spot; B, canker; C, wilt.

Many symptoms of plant diseases involve the death of plant tissues. Leaf spots are an accumulation of **necrotic** (dead) cells. Some diseases cause leaf spots that expand to form a **blotch**. Some diseases cause large areas of leaves or flowers to die; necrosis affecting large areas of leaves or flowers is called **blight**. Some diseases cause necrosis that is restricted to the edges of leaves, a symptom known as **scorch**. Stem lesions that are sunken are called **cankers** (Figure 1.4B), although some cankers may be raised by the production of callus tissue by the plant. Stems, roots, wood, fruits, and storage parts such as tubers, corms, and bulbs are subject to **rots**. There are **soft rots**, in which tissues liquefy and collapse, and **dry rots**, which may result in **mummification** of tissues such as fruits and tubers. Woody plants, as part of their defense against infection by pathogens, may produce visible secretions of liquids. Depending on the consistency of the liquid, this symptom is called

bleeding, gummosis, or **resinosis**. Germinating seeds and seedlings are particularly vulnerable to disease. Diseases that cause seedlings to fail to emerge or to topple over at the soil surface as a result of damage that weakens the stem are referred to as **damping-off**.

Not all symptoms involve necrosis. Many symptoms consist of changes in the coloration or form of plants or plant parts. **Mosaic, mottle, streak, stripe**, and **yellowing** (**chlorosis**) are changes in color. **Rugosity, leaf roll, galls**, and **witches' brooms** are changes in form. Some of these symptoms are caused by pathogens that affect the normal balance of growth regulators in certain plant tissues.

Wilt is a response to the disruption of the transport of water in the plant due to invasion of the xylem or roots by a pathogen (Figure 1.4C). Over time, diminished water transport will affect the growth of the plant, and the entire plant may be stunted. If the phloem is affected, yellowing (chlorosis) or other variations in color may occur in leaves, but the leaves will not generally appear wilted. Chlorosis may be due to a phloem pathogen or a nutrient deficiency. Dying plant tissues also may turn yellow. When symptoms such as stunting or chlorosis affect an entire plant, one must consider which functions may be impaired to determine the source of the problem.

Many of the symptoms described above affect individual plants, but disease symptoms may also be exhibited by groups of plants. For example, the symptoms of many turfgrass diseases, such as dollar spot and brown patch, involve groups of plants.

When it can be determined that a disease is affecting a plant species or closely related plants in an area, try to discern patterns of disease in the plant population. Are diseased plants randomly scattered, or are they localized in concentrated areas (disease foci)? Are they concentrated along the borders or edges of a planted area, near plant barriers, or near bodies of water? Are all or nearly all plants affected equally? Airborne pathogens tend to affect plants uniformly, but soilborne pathogens tend affect plants in somewhat circular patches ("hot spots"), surrounded by relatively healthy plants (Figure 1.5).

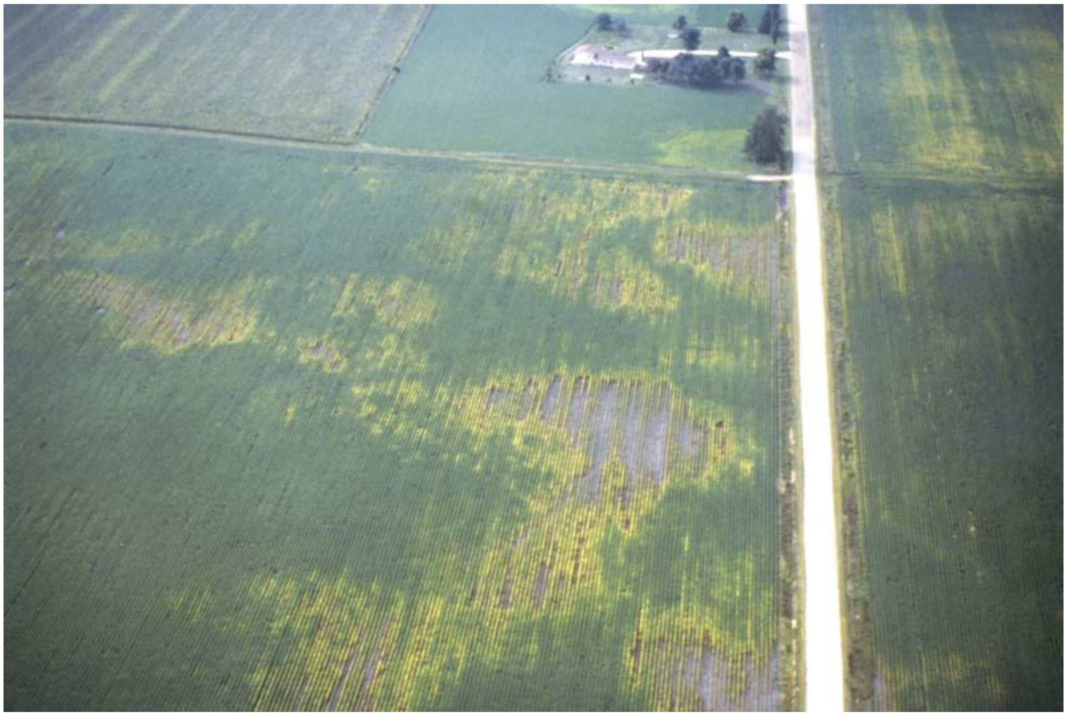


Figure 1.5. Circular pattern of damage caused by the soybean cyst nematode. Such patterns are typical of diseases caused by soilborne pathogens.

In addition to noting symptoms, it is important to accurately identify the species and sometimes the cultivar (variety) of affected plants. Common names of plants vary from one region to another, and the same name may be used for entirely different species in different locations. For example, cedar-apple rust is a disease of red-cedar (juniper), not white-cedar (also known as arborvitae). Once you know what a healthy plant should look like, you can determine how the affected plant is expressing its disease.

The **host range** of a pathogen is the set of species in which the pathogen can cause disease. In diagnosis, it is important to note whether a disease is restricted to a single plant species or cultivar or whether it occurs in different species of the same genus or in plants belonging to the same family. In some cases, even unrelated plants may appear to be affected, which can be an important diagnostic clue.

Symptoms alone are not enough for accurate diagnosis of many plant diseases.

The Pathogen

The second vertex of the plant disease triangle is the pathogen. The word *pathogen* comes from the root word *pathos* (“suffering”) and the suffix *-gen* (“origin,” or “genesis”). Together they mean “the cause of a disease.”

Most plant diseases are caused by **parasites**. A parasite is an organism that obtains its nutrients from another living organism. All living organisms have parasites. Parasites of plants include fungi, bacteria, nematodes, viruses, and even other plants.

Parasites become pathogens when they do not merely live on and obtain nutrients from a host organism but actually cause harm to the host, resulting in disease symptoms. Some parasites that are relatively harmless in their natural environment can become serious pathogens in agricultural settings in which crops are grown in monoculture (plantings of many individual plants of the same species). Exotic pathogens are especially dangerous when they are introduced into new environments in which plants have no natural resistance to them.

Pathogens that are also parasites are **infectious**. They can be spread (**transmitted**) from plant to plant, potentially causing an epidemic. These living organisms that cause diseases are sometimes called **biotic pathogens**.

There are also **abiotic** (nonliving) agents that cause disease. **Noninfectious** factors may affect many plants in an area, but the diseases they cause are not “contagious,” i.e., they are not transmitted from one plant to another. In humans, noninfectious diseases include arthritis and iron-deficiency anemia, in contrast to infectious diseases, such the common cold and smallpox. In plants, noninfectious diseases are caused by abiotic factors such as air pollution, low temperatures, and nutrient deficiencies.

Parasite: derives its food from a living host
Pathogen: causes disease in a living host
Biotic pathogens:

- infectious
- transmissible

Abiotic factors:

- noninfectious
- nontransmissible

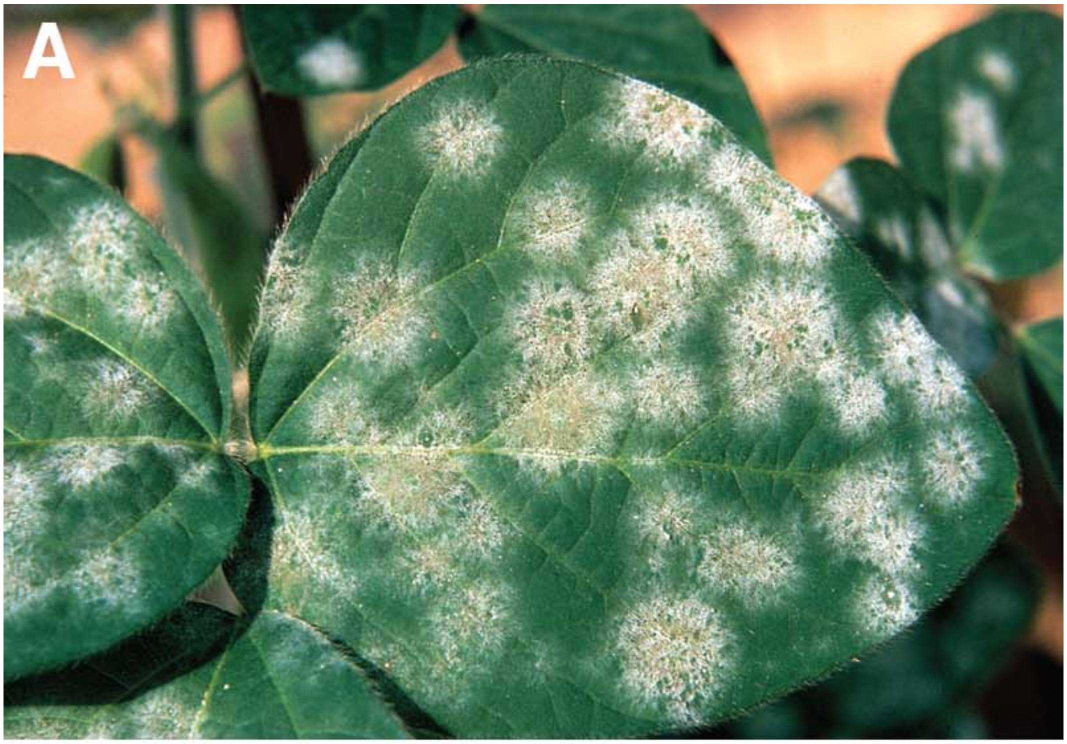
There are clues that help to determine whether a disease is infectious or noninfectious. Biotic pathogens often produce **signs**—pathogen structures or products on or in diseased plants (Figure 1.6). Some signs are visible to the unaided eye, and others can be viewed only with a hand lens or microscope. Some signs, such as those of viruses, are so small that they can be seen only under the high magnification of an electron microscope.

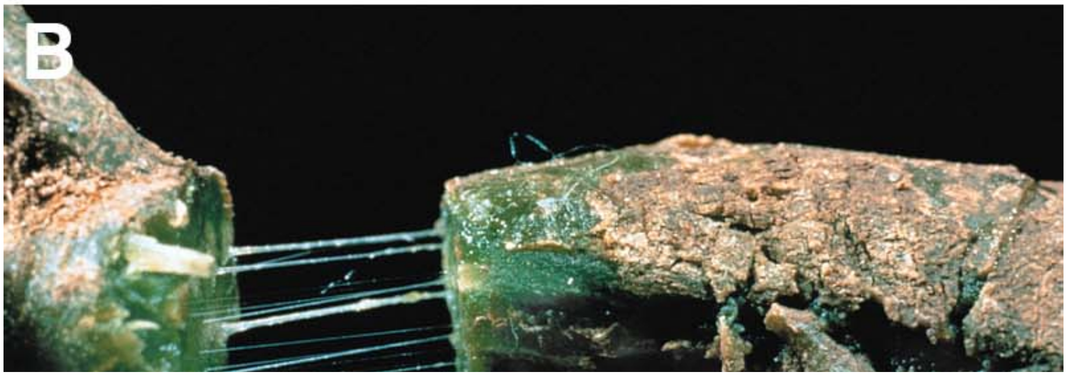
Symptoms are the expression of disease by a plant as a response to a pathogen.
Signs are structures or products of a pathogen on or in diseased plants.

CASE STUDY: APPLE SCAB

Signs

Venturia inaequalis, the fungus causing apple scab, produces spores that turn leaf lesions velvety olive green in the early stages of the disease. These spores initiate new infections of leaves and developing fruit. Sexual fruiting bodies of the fungus are produced in fallen leaves in the early spring. These structures are tiny but can be observed with a microscope.





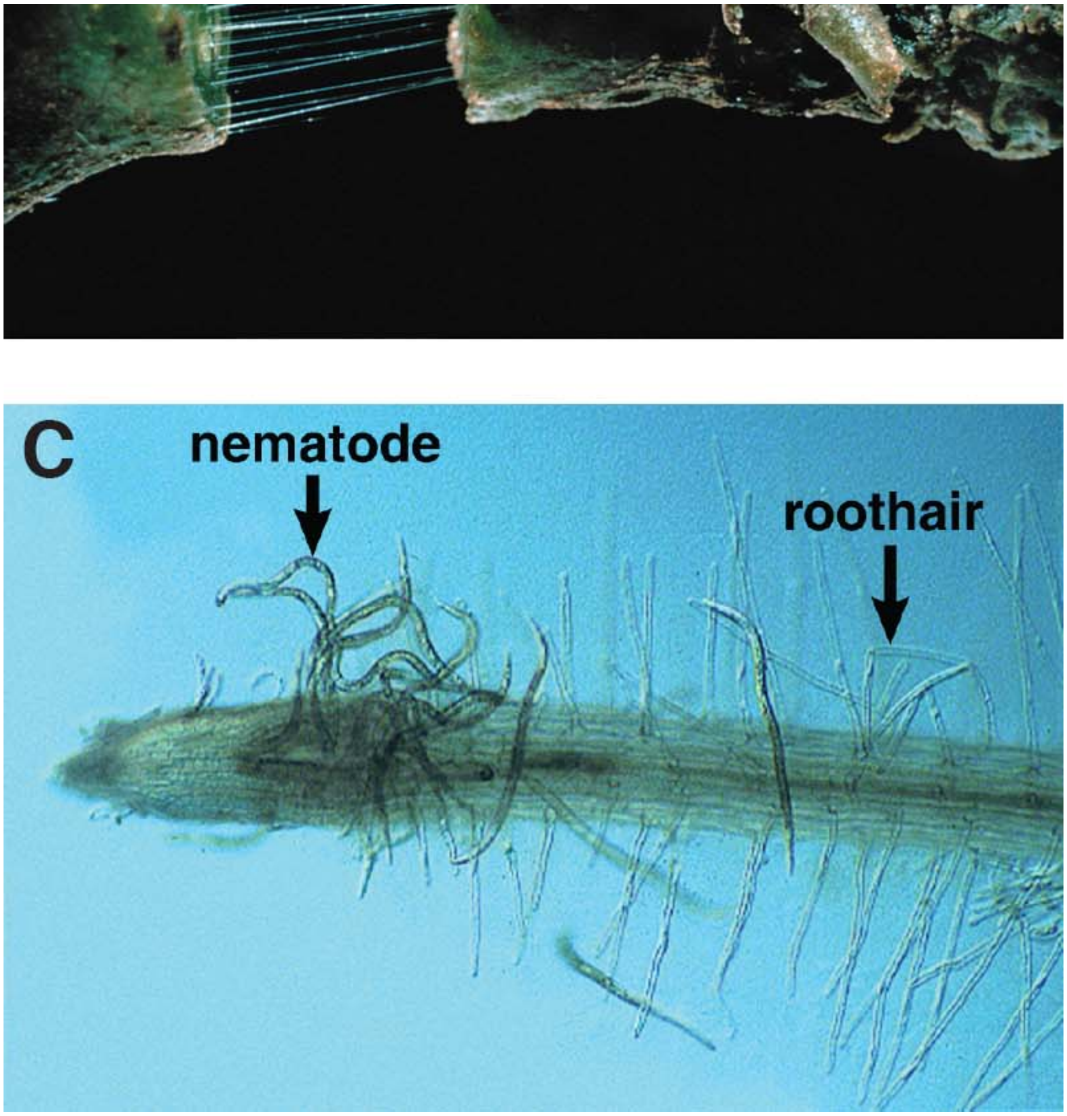


Figure 1.6. Common signs of pathogens: A, mycelium and spores of a powdery mildew fungus; B, bacterial ooze; C, nematodes on a root.

Signs of some pathogens are easier to observe than others. For example, parasitic plants, such as mistletoe, are easily seen and diagnosed. Fungi produce various signs, from mushrooms to **spores** and **fruiting bodies** small enough that magnification is required for viewing them. Some diseases are named for the distinctive signs produced by the pathogens that cause them: orange, powdery spores are characteristic of rust diseases, and black, powdery spores are characteristic of smut diseases. When bacteria accumulate, they may produce a visible **ooze**. Viruses are too small to be seen with a light microscope, but certain structures (inclusion bodies) produced in virus-infected tissues can sometimes be observed. Nematodes are small worms found in soil and roots, from which they must be extracted in a laboratory for observation. Signs of specific pathogens are described in the chapters on the various types of pathogens (Chapters 2–6).

There are no signs of abiotic agents that cause plant diseases. However, there may be other clues that a disorder is abiotic. Many abiotic factors harm various kinds of plants and may affect all plant species in an area. Biotic pathogens tend to be more host specific, affecting a single species or even members of a whole plant family, but not all of the plants species in an area. Symptoms of an abiotic disorder are often at a similar stage of development throughout an area. In contrast, symptoms of a disease caused by a biotic pathogen may be in various stages in different plants at the same time; severely diseased plants may be present along with newly infected plants, or new lesions may be present on a plant along with older areas of infection.

If necrotic tissue is present, there is likely to be a sharp border between the healthy and the diseased tissue affected by an abiotic disorder such as herbicide injury. When a leaf is infected by a biotic pathogen, a halo of yellow to red to purple dying cells is often formed at the margin between healthy and dead cells.

It is important to remember that tissue that is dead or dying as a result of abiotic factors may be quickly invaded by secondary organisms that were not the cause of the initial disease. Further investigation is required to identify the primary disease agent.

Biotic pathogens often produce signs, which are evidence of their presence and can aid in diagnosis. There are no signs of abiotic factors.

The Environment

The environment in which plant disease occurs consists of a wide range of factors that should be investigated when a diagnosis is being made. These factors include recent temperatures (such as extreme highs and lows), rainfall or irrigation (amounts, timing, and source), and light intensity or shade. Characteristics of the soil, such as drainage, soil type, and pH, are also important. In surrounding areas, are there surface waters, such as ponds or streams? Do hedgerows or landscape plantings impede airflow? What is the general direction of airflow, which may carry spores? Are **vectors** (organisms such as insects that help spread pathogens) present?

CASE STUDY: APPLE SCAB

The Environment

The fungal spores that cause the initial infection are released in the spring, between the time when apple leaves begin to expand and the time when the flower petals fall off. Temperatures between 15 and 22°C (60–72°F) are optimal for infection. Leaves, flowers, and fruit must be wet for at least 9 hours for spores to germinate and initiate infection. The disease is more severe in years with repeated rain in the spring. Little apple scab is observed when the weather is hot and dry.

Human activities introduce other environmental factors. What types and amounts of fertilizer have been applied recently? Have other chemicals, such as herbicides, insecticides, or fungicides, been applied to the affected plants or nearby plants in the past few weeks? What other plants are growing in the area? In plantings of annuals, what was grown in the previous season? In greenhouses, what is the source of the planting medium, and what type of medium is used?

► **DID YOU KNOW?**

People can alter the susceptibility of plants to diseases. For example, hybrid corn lines introduced into corn seed production in the 1960s produced plants that were highly susceptible to race T of the leaf spot fungus *Bipolaris maydis*, which caused losses of over $1 billion in the United States in 1970.

► **DID YOU KNOW?**

After the accidental worldwide distribution of new strains of the late blight pathogen, potato losses due to late blight in the United States approached $1 billion in 1992. Losses in developing countries are about $3 billion annually. Late blight continues to cause the loss of about 25% of the world's potato yield every year.

An environment favorable for disease development—the third vertex of the disease triangle—consists of factors affecting the plant, factors affecting the pathogen, and sometimes additional organisms, such as vectors.

How do we know what organism causes a disease?

Can disease be diagnosed by comparing a diseased plant with photographs of diseased plants? This approach may be misleading, because it focuses on only one component of the disease triangle: the susceptible plant and its symptoms. Many diseases can cause the same or similar symptoms. In human health, for example, a sore throat can be the result of an allergy, a viral infection, or shouting too long at a ball game. In addition to studying the symptoms of diseased plants, diagnosis requires consideration of the other two components of the disease triangle—the pathogen and the environment.

If fungal growth or some other sign is observed on a diseased plant, can one conclude that the fungus is the cause of the disease? This, too, may be misleading. Once dead or dying tissue is present, it is common for secondary invaders such as **saprophytes** (organisms that obtain their nutrients from dead organic matter) to **colonize** the tissue. How can we identify the primary pathogen?

Shortly after the germ theory became accepted among scientists, it became clear that some method was necessary to prove the **pathogenicity** (the ability to cause disease) of an organism found associated with diseased tissues. Robert Koch, an early microbiologist, first accomplished this in studying anthrax, a disease of warm-blooded animals. Koch isolated bacteria from the blood of sheep and cows with anthrax and then inoculated rabbits, mice, and sheep with the isolated bacteria. When the inoculated animals subsequently became sick with anthrax, he concluded that the bacteria caused the disease. His method for establishing proof of pathogenicity, known as Koch's postulates, has been adopted by scientists who study human and animal diseases and by plant pathologists as well. Koch's method originally included only the first three steps; the fourth step was added later.

► DID YOU KNOW?

Robert Koch (1843–1910) began studying anthrax in Germany around 1873, during a severe epidemic that killed many sheep and cows and even some humans. The disease is caused by a spore-forming bacterium, *Bacillus anthracis*. It is one of the largest bacteria known, so it could be more easily observed than most other microbes with the microscopes in use during Koch's time. Koch used wild field mice caught in his barn as experimental animals. He discovered that the bacterium could be isolated in pure culture in the aqueous humor of rabbit eyes. He later used cow eyes, which were easily available from slaughterhouses. Eventually he grew pure cultures on nutrient media with gelatin. He drew important public health conclusions about the dangers of spore-filled blood and tissue of diseased animals and cadavers. Koch later made careful epidemiological studies of cholera, another bacterial disease, which led to public health practices such as water filtration to improve the safety of urban water supplies.

Koch's Postulates for Proof of Pathogenicity

1. The suspected pathogen must be consistently associated with diseased plants.
2. The suspected pathogen must be isolated in a pure culture and its characteristics noted.
3. The disease must be reproduced in a healthy plant inoculated with the isolated organism.
4. The same pathogen characterized in step 2 must be isolated from the inoculated plant.

There are some difficulties in applying Koch's postulates. For example, modifications have been made over time to accommodate pathogens that cannot be grown in culture (Postulate 2). Also, recreating a disease requires all three vertices of the disease triangle (Postulate 3). Sometimes a plant must be at a certain stage of growth to be susceptible or in a certain environment for disease to develop. Nevertheless, fulfilling Koch's postulates, with modifications as needed, remains the accepted method of proving that a particular organism causes a given disease. When reports of new plant diseases are published, they often note explicitly that Koch's postulates have been fulfilled or describe the process used to fulfill them. Although Koch's postulates are the guidelines still used today for new diseases of plants and animals, they are not used in routine diagnosis of well-known diseases.

Koch's postulates are used to prove that an organism associated with diseased tissue is the cause of the disease.

Once the pathogen causing a particular disease has been confirmed by Koch's postulates, a disease triangle can be constructed for use in routine diagnosis and disease management. In diagnosis, the triangle offers the key points for investigation. A susceptible plant can be growing in an environment that is favorable for a particular disease but will remain healthy unless the pathogen is also present. Even when a susceptible plant is exposed to a virulent pathogen, no disease develops if environmental conditions are not favorable for disease development.

Using the Disease Triangle in Diagnosis

Step 1. The Plant. Identify the species and cultivar of the plant, when possible. Determine what a healthy specimen should look like, so that you can recognize abnormalities of the plant in question. Carefully observe aboveground symptoms and also belowground symptoms, if necessary. In addition to observing symptoms expressed by a single plant, look for disease patterns in the plant population. Note the apparent host range (i.e., what plant species are affected).

Step 2. The Pathogen. Look for signs, evidence of a pathogen or its parts, both above ground and below ground, if necessary. A hand lens is often helpful, but laboratory evaluation is commonly necessary to check for signs. Signs are often more obvious under moist conditions (for example, before dew has dried in the early morning or after rain or irrigation). Abiotic factors produce no signs, and there may be no visible signs of some biotic pathogens, such as viruses. Abiotic factors may affect nearly every plant species in an area to some degree, but biotic pathogens tend to be restricted to a single species or members of the same family of plants. Some signs may be evidence of secondary invaders, which are not the initial cause of the disease.

Step 3. The Environment. Disease will develop in susceptible plants in the presence of a pathogen only if the environment is favorable. Several environmental factors are important, including natural factors and those imposed by human activities. Determine the recent conditions for plant growth, including chemical and fertilizer applications, characteristics of the soil or growing medium, and environmental factors, such as temperature and water supply. Look for factors that commonly affect disease development, such as the timing and amount of irrigation or rainfall, air movement, and the nutrient status of the plant.

CASE STUDY: APPLE SCAB

Koch's Postulates

Koch's postulates do not need to be completed for routine diagnosis of well-known diseases, but they were completed for apple scab in the early years of plant pathology. The fungus that causes apple scab will grow on potato-dextrose agar, so it can be isolated from diseased tissue. Only expanding tissue of leaves, flowers, and fruit is susceptible to infection, and therefore the disease can be reproduced in healthy trees inoculated with the isolated pathogen only if they are inoculated at the appropriate stage of development.

The disease triangle is also used to design approaches to management of plant diseases. Practices that affect the plant, the pathogen, or the environment may be useful in managing plant diseases. Resistance genes or planting methods can make plants less susceptible. Various methods can reduce pathogen populations or restrict the exposure of plants to pathogens. The environment can be manipulated so that it is less favorable for disease development. The effectiveness of management practices depends on the type of plant, the type of pathogen, and the environment in which the plant is grown.

What are biotrophs and necrotrophs?

Plant diseases often involve the interaction of plants and parasites. Thus, diseases are greatly affected by the biology of the parasites.

Some parasites are so dependent on their hosts that they are unable to exist without them. These parasites, called **biotrophs**, or **obligate parasites**, can obtain nutrients only from living plant cells. They often form some kind of survival structure in the absence of a host. In some cases, biotrophs do not form a survival structure and will perish if they do not find a way to move to another living plant before the host plant dies.

Most plant pathogens are more flexible in their adaptation and can live either as **parasites** or as **saprophytes** (**saprobes, saprotrophs**), organisms that obtain nutrients from dead organic matter. Organisms with the flexibility to switch back and forth between the parasitic and the saprophytic life style are described as **facultative**. **Facultative saprophytes** are better adapted to living as parasites but can survive as saprophytes when necessary. **Facultative parasites** are primarily saprophytes but can live as parasites if given the opportunity to invade compromised or senescent plant tissues. Thus, pathogens can be placed along a spectrum from completely obligate parasites to facultative saprophytes to facultative parasites. Many other bacteria and fungi are **obligate saprophytes**, living only on dead organic matter and incapable of causing plant disease. These organisms may act as secondary invaders of dead and dying tissues. They contribute to the decay of plants and other organisms.

Parasites live in or on another living organism and obtain nutrients (food) from it.
Saprophytes obtain nutrients from dead organic matter.

The lifestyle of the parasite helps determine the kind of disease that will occur. Because biotrophs require living host cells for survival, they are relatively delicate in their invasion of host tissues, at least in the early stages. Fungi in this group may produce **haustoria**, specialized structures that permit them to absorb nutrients without killing host cells immediately (Figure 1.7). To penetrate plant tissue, biotrophs tend to invade through natural openings, such as stomata, or directly into cells (e.g., viruses can be transmitted by aphids or other insects with piercing-sucking mouthparts). Biotrophs do not produce toxins (poisons) or enzymes that rapidly destroy host cells. They are generally capable of invading host plants that are at any stage of development. A biotroph is typically well adapted to be a parasite of a single plant species or closely related species.

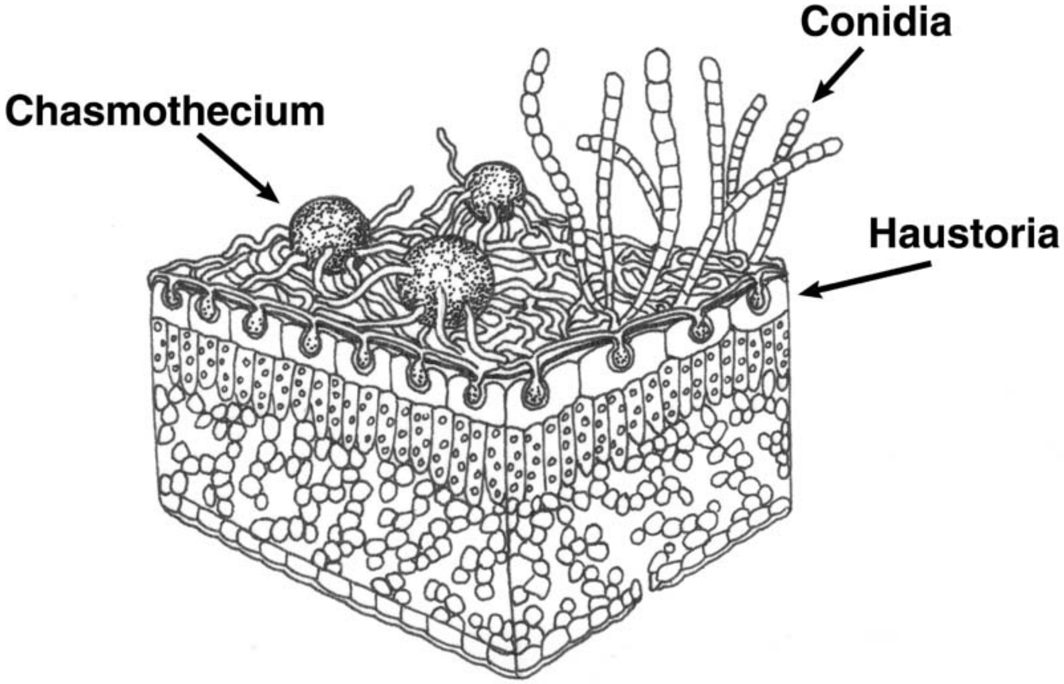


Figure 1.7. A powdery mildew fungus, a biotroph, on a leaf surface.

At the other end of the spectrum of parasitism are the facultative parasites and facultative saprophytes, or **necrotrophs**. As the name suggests, they obtain nutrients from dead organic matter or from dead or dying cells of living plants. In contrast to the more delicate invasion by biotrophs, necrotrophs tend to produce toxins and enzymes that rapidly kill and degrade plant tissue and inhibit defense mechanisms by the plant. Necrotrophs commonly penetrate plants through wounds or natural openings. Weaker cells are most vulnerable, such as those of very young tissues and those of old, senescing tissues. Because these parasites feed on weakened, dying, or dead cells, they are not as specialized in their choice of hosts and have wider host ranges than biotrophs.

Biotrophs (obligate parasites)	**Necrotrophs (facultative parasites and facultative saprophytes)**
▸ have narrow host ranges	▸ have wide host ranges
▸ cannot grow as saprophytes	▸ can grow as saprophytes
▸ attack healthy host tissue at any stage	▸ attack young, weak, or senescent tissues
▸ kill host cells slowly	▸ kill host cells rapidly by producing toxins or enzymes
▸ penetrate directly or via natural openings	▸ penetrate through wounds or natural openings

CASE STUDY: APPLE SCAB

Biotroph or Necrotroph?

The apple scab fungus, *Venturia inaequalis*, is a necrotroph. It is a parasitic fungus that predominantly invades the superficial tissues of leaves, flowers, and fruit, causing necrosis and inducing a defensive reaction in which the plant forms cork cells in affected tissue. Later, the fungus survives the winter as a saprophyte in fallen leaves.

The symptoms of a plant disease can suggest the type of relationship between the host and the pathogen. A diseased plant that is stunted or distorted but remains relatively green is probably infected by a biotroph. Nematodes, powdery mildew fungi, and viruses are examples of biotrophs causing plant diseases. A diseased plant with a large amount of dead and dying tissue is probably infected by a necrotroph. Most bacteria and many fungi that cause plant diseases are necrotrophs.

Common Diseases Caused by Biotrophs

- nematode diseases
- phytoplasma diseases
- virus diseases
- downy mildews
- powdery mildews
- rusts

Common Diseases Caused by Necrotrophs

- anthracnoses
- cankers
- fruit rots
- leaf spots and blights
- root rots
- vascular wilts

Just as biotrophs and necrotrophs vary in the symptoms they cause, they also have variable survival mechanisms. Necrotrophs are capable of living as saprophytes in the absence of plants. Both necrotrophs and biotrophs may produce survival structures to remain viable until a new host plant is nearby. Both necrotrophs and the survival structures of bio-trophs may persist in soil, on planting parts (such as bulbs and tubers), in plant debris at the end of a growing season, on dirty pots and tools, and on seeds. Some biotrophs also pass from one plant to another by infection of seeds attached to the mother plant or during vegetative propagation, and some are spread by vectors (e.g., insects or nematodes that feed on plants). Thus, new plants can become infected by pathogens from diseased plants that existed previously.

Biotic pathogens can be divided into two categories (biotrophs and necrotrophs) that describe their approach to causing disease. Biotrophs require living plant tissues. Necrotrophs usually produce destructive toxins and enzymes that destroy plant tissues.

What are disease cycles and how can we use them?

Pathogens, like their host plants, have life cycles. These may be as simple as that of a bacterium, in which a single cell divides into two new cells. Rust fungi have complex life cycles that may include up to five spore stages that require two unrelated hosts. Plants, too, have variable life cycles that may affect their interaction with pathogens. When interactions between a plant and a pathogen result in disease, the interactions are described by the **disease cycle** (Figure 1.8).

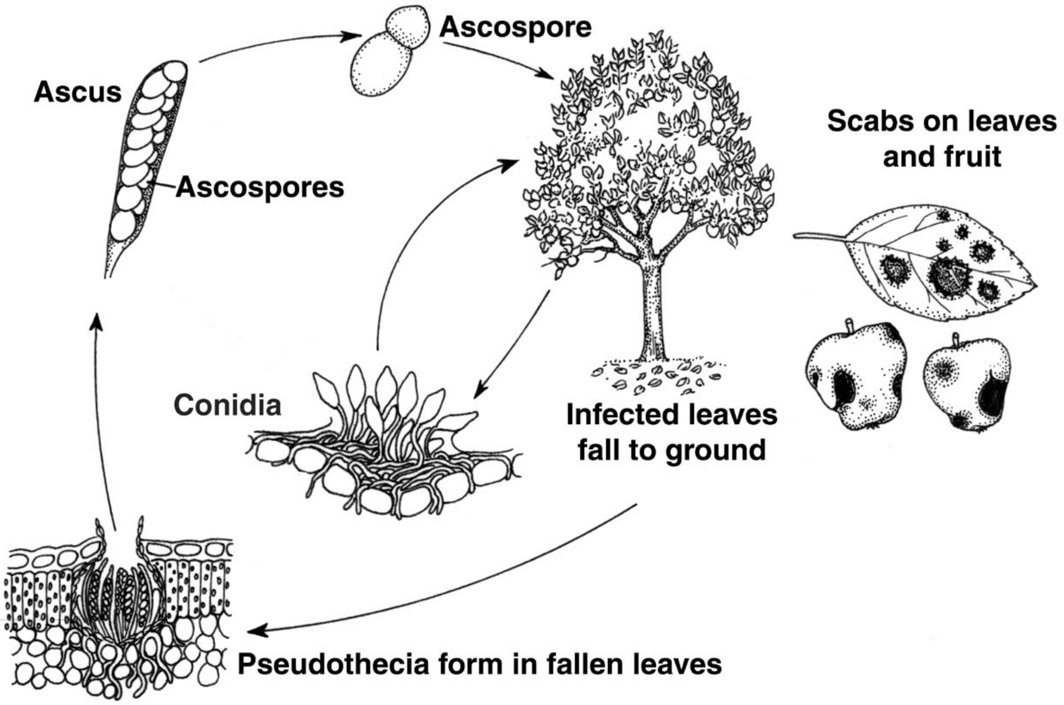

Ascospore
Ascus
Ascospores
Scabs on leaves and fruit
Conidia
Infected leaves fall to ground
Pseudothecia form in fallen leaves

Figure 1.8. Disease cycle of apple scab.

A disease cycle may be simple or complex, but all disease cycles follow a pattern of discrete steps occurring in a predictable order. Disease cycles may be completed in as little as a few days or (in some tree diseases) as long as several years. It is important to be able to identify the stages of the disease cycle of each disease, because they will suggest ways to prevent or manage the disease.

Primary Inoculum

The structure or part of the pathogen that initiates disease is called the **primary inoculum** (or **initial inoculum**). Finding and eliminating sources of primary inoculum are important for disease management. The primary inoculum may or may not be the same structure that serves as the survival stage of the pathogen. In some cases, a pathogen in its survival stage must produce a new structure, such as an airborne spore, to serve as primary inoculum.

Dispersal of Inoculum

Because plants do not move on their own, many plant pathogens require some means of moving from one plant to the next (Figure 1.9). Fungi produce many kinds of spores and use a variety of mechanisms to disperse them. Some spores are airborne and are even forcibly discharged into the air, but others are sticky and rely on water, tools, or passing insects for dispersal. Similarly, bacteria tend to occur in sticky masses that are dispersed by water, tools, or insects. Other pathogens may be spread during vegetative propagation or rely on movement by vectors, such as insects that acquire and introduce pathogens to plants when feeding. By producing and distributing seeds and other propagative plant parts, humans have inadvertently moved various pathogens around the world and, in some cases, introduced pathogens into new crop regions.

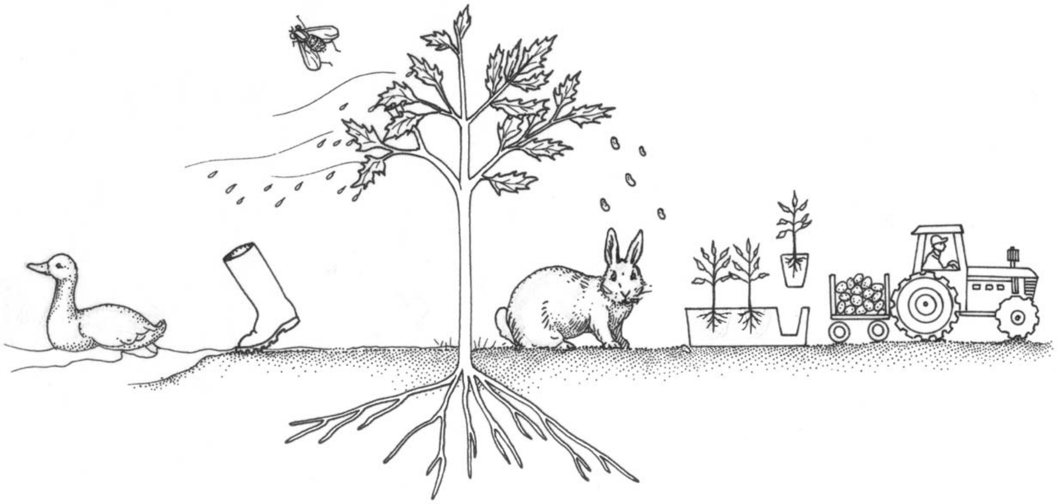


Figure 1.9. Some common means of pathogen dispersal: insect vectors, tools and equipment, vegetative propagation, wind, and water.

The production of inoculum of some pathogens must be coordinated with the stage of plant development. Spores produced in the spring must land on (**inoculate**) a plant that is in the appropriate stage to become infected. For example, spores produced before a tree has grown leaves will fail to successfully establish an infection. Inoculum must also reach a part of the plant that can be invaded, known as an **infection court**.

Infection Courts

Where does an infection start? Only certain parts of plants, called **infection courts**, are susceptible to particular pathogens. For example, the bark of a tree prevents most pathogens from invading the living tissues below it. Pathogens that infect woody plant parts usually rely on wounds to gain entry. Roots are most vulnerable near the root cap or where lateral roots break through the cortical tissues. The soft tissues of leaves, fruits, and flowers are particularly susceptible to invasion by various pathogens. Pathogens that land on plant tissue must find an entry point, such as a wound or a stoma (**indirect penetration**), or else they must create an entry point, (**direct penetration**).

CASE STUDY: APPLE SCAB

Primary Inoculum, Dispersal, and Infection Courts

The primary inoculum of the apple scab fungus, *Venturia inaequalis*, is ascospores, which are forcibly discharged from fruiting bodies produced in fallen leaves from the previous season. Air currents carry these tiny spores and can transport them to infection courts on expanding tissues of leaves, flowers, and fruit. Under favorable environmental conditions, a small proportion of the ascospores will successfully infect plant tissue.

Infection and Colonization

The ultimate goal of most pathogens is to establish a relationship in which it acquires food from the host plant. This is a complex process requiring recognition of the host plant and the ability to overcome defense mechanisms that the plant may employ. The pathogen must invade or penetrate the host plant and establish a parasitic relationship with it to complete **infection**. Penetration does not necessarily result in infection. Only after successful infection can the pathogen begin to **colonize** the plant tissue. How the pathogen colonizes the plant is dependent on its specific characteristics. Following penetration, there is an **incubation period** during which no symptoms are apparent. This is one of the most important differences between diseases and insect problems. By the time disease symptoms are noticeable, the pathogen has established an infection and colonized enough plant tissue to cause visible symptoms. It may already be impossible to stop the disease from further development. Even systemic fungicides that can enter plant tissues to halt very early infections by fungi may be of little use once significant disease symptoms are visible.

Secondary Inoculum

A successful parasitic relationship requires that the pathogen sustain itself and produce new inoculum. No host plant will survive forever, so pathogens must eventually produce inoculum to infect a new host plant. Inoculum produced on an infected plant is considered **secondary inoculum** if it functions to establish new infections during the growing season in which it is produced. These infections may be on the same plant, on neighboring plants, or on distant plants.

The secondary inoculum may or may not be identical to the primary inoculum. Secondary inoculum produced by bacteria, nematodes, and viruses is identical to the primary inoculum of these pathogens. In contrast, the spores that initiate a fungal infection may be very different from the spores produced for secondary infections during a growing season. Thus, dispersal mechanisms may be different for primary inoculum and secondary inoculum. Some pathogens do not produce any secondary inoculum during a growing season.

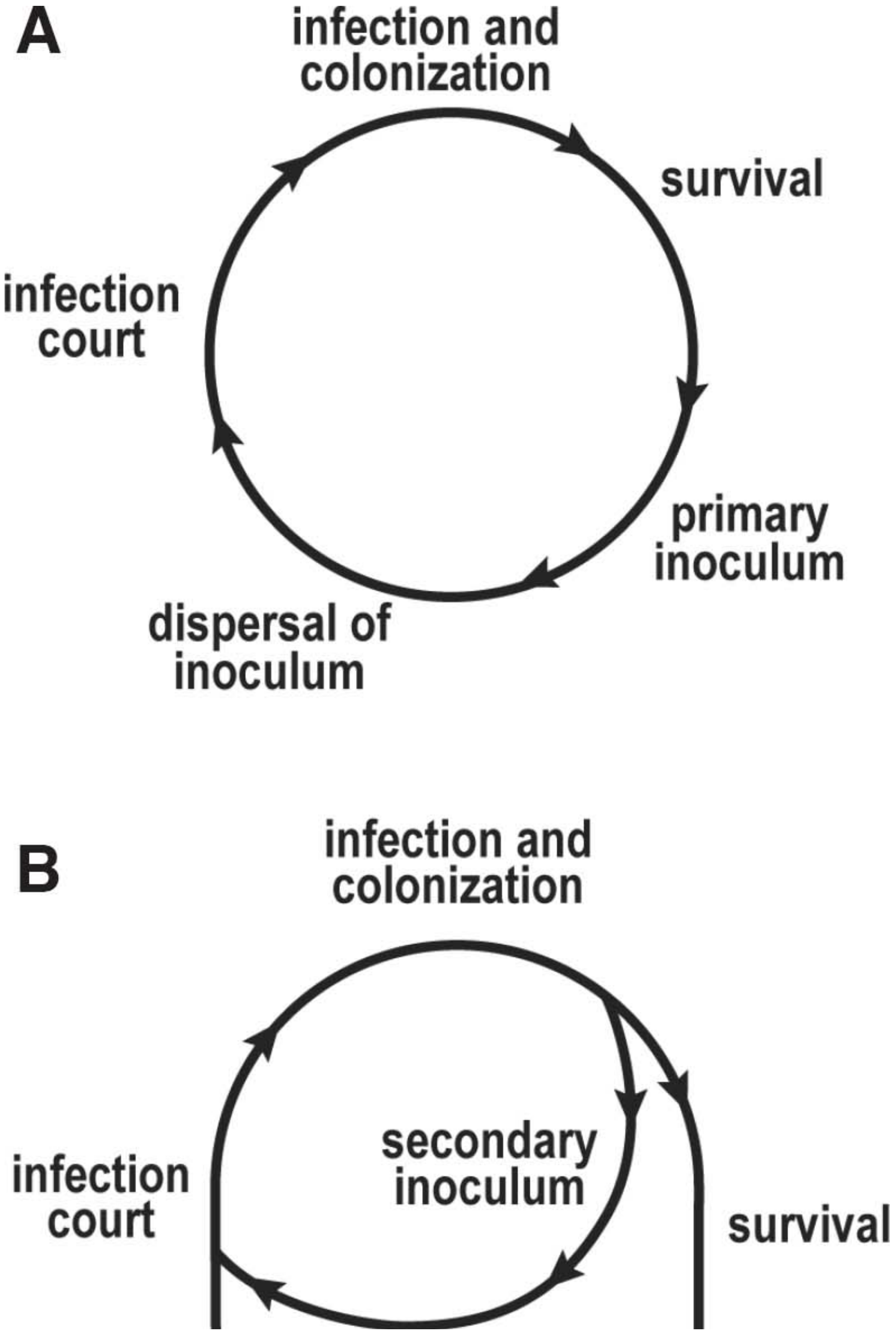
► DID YOU KNOW?

A single pustule of the coffee rust fungus can produce 150,000 spores, which serve as both primary and secondary inoculum.

► DID YOU KNOW?

The Great Bengal Famine caused the death of 2 million people in India in 1943. Rainy weather during an unusually long monsoon season favored epidemics of a leaf spot disease caused by the fungus *Bipolaris oryzae*, which destroyed the rice crop. This disaster occurred during the Second World War when India was still a British colony. With the Japanese army occupying neighboring Burma (Myanmar), India had no access to supplies from rice-growing regions of southeast Asia, and the Bengali people starved.

When infection is caused only by primary inoculum, there is a single disease cycle during a growing season. Such diseases are **monocyclic** (Fig. 1.10A). A common example is Verticillium wilt, a vascular wilt (xylem) disease caused by a fungus that enters plants through their roots. Tiny black survival structures (microsclerotia) in the soil serve as the primary inoculum. After infection, the fungus remains in xylem tissue until the plant dies. It then enters a saprophytic growth stage and eventually produces new microsclerotia, which are released into the soil as the plant tissue decays. The microsclerotia serve as primary inoculum in the next growing season.


A
infection and colonization
survival
primary inoculum
dispersal of inoculum
infection court
B
infection and colonization
secondary inoculum
survival
infection court

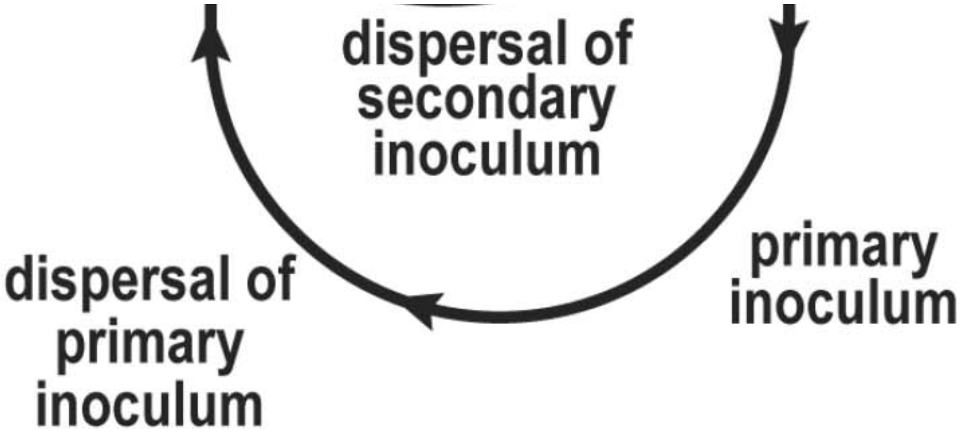

dispersal of secondary inoculum
primary inoculum
dispersal of primary inoculum

Figure 1.10. A, Monocyclic disease. B, Polycyclic disease.

Monocyclic Diseases

- Dutch elm disease
- stinking smut
- Verticillium wilt

In other diseases, several to many generations of secondary inoculum may be produced, resulting in widespread disease development. When secondary inoculum is produced, the potential for secondary disease cycles exists and a **polycyclic** epidemic (Fig. 1.10B) may result. Look back at the disease cycle shown in Figure 1.8 to see how it works.

Polycyclic Diseases

- apple scab
- coffee rust
- late blight of potato and tomato

The time between infection by primary inoculum and the production of secondary inoculum is called the **latent period**. The terms *incubation period* and *latent period* have sometimes been used in confusing ways. However, it is important to differentiate between the interval from infection until the appearance of symptoms (incubation period) and the interval from infection until the production of secondary inoculum (latent period). Sometimes these intervals coincide, but they may be separated by several days or more.

CASE STUDY: APPLE SCAB

Infection, Colonization, and Secondary Inoculum

Unlike many fungal parasites, *Venturia inaequalis* invades leaves and fruit only superficially, between the cuticle and the epidermis. From 9 to 30 days after infection by ascospores, the primary lesions produce conidia, the secondary inoculum, which are dispersed by water. Up to 100,000 conidia can be produced by a single lesion. Apple scab is a polycyclic disease. The number of generations of conidia produced each year depends on environmental conditions and the genetic resistance of the tree on which they are produced.

Survival of Pathogens

Annual plants die naturally at the end of the growing season or may die prematurely from disease or other causes. Pathogens of these plants must either produce survival structures or begin to function as saprophytes. Perennial plants may continue to harbor pathogens for years until their demise. But no plant is immortal, so even pathogens of perennial plants must find a new plant host eventually. The survival stage, when a pathogen is separated from its host, is a particularly vulnerable time for most biotic pathogens. This stage is very important because it is usually the source of primary inoculum that will lead to new disease outbreaks.

CASE STUDY: APPLE SCAB

Survival of the Pathogen

In living leaves, the apple scab fungus is confined to the area between the host cuticle and the epidermis. Once infected leaves have fallen, the fungal mycelium colonizes them completely as a saprophyte. *Venturia inaequalis* usually overwinters in fallen leaves, surviving as mycelium and as early stages of sexual fruiting bodies (pseudothecia). These structures are the source of ascospores in the spring. Therefore, removal and destruction of fallen leaves is an effective way to reduce the disease. If leaves cannot be completely removed, as in a commercial orchard, protection of new leaves with fungicides, beginning at the time of bud break, is necessary in humid climates.

Disease cycles of biotic pathogens begin with primary inoculum that is dispersed to an infection court. If a disease is polycyclic, secondary inoculum will be produced, and additional infections may occur on the same host plant or other host plants during the growing season. The survival stage is often the source of primary inoculum that will initiate a new disease cycle.

Disease Management Using the Disease Cycle

Management of a plant disease can focus on any of the important steps in the disease cycle: primary inoculum, infection courts, secondary inoculum, the dispersal of primary and secondary inoculum, and the survival stage.

Environmental factors affect plant diseases in all stages of the disease cycle. They influence

- plant development and the ability of the plant to mount defenses against invasion,
- dispersal of inoculum, both primary and secondary,
- the ability of the parasite to penetrate the plant, and
- the survival of the parasite in the absence of the host plant.

Water is a major environmental factor in disease, because it allows parasites to move to infection courts and penetrate host tissue. Fungal spores landing on a plant surface commonly require water for germination and the production of infection structures. Zoospores are motile and can swim to plants or on plant surfaces. Nematodes can move toward plants and on plant surfaces in a thin film of water. Bacteria require water for multiplication and infection. Seeds of parasitic plants require water for germination.

Approaches to disease management can be divided into four general categories: avoidance, exclusion, eradication, and protection. **Avoidance** of disease involves choosing a planting location where the pathogen is not present or choosing a planting time when the pathogen is not active. **Exclusion** of disease involves legal restrictions, such as quarantines, or the production of pathogen-free seed or propagative parts, so plants can be grown in the absence of the pathogen. **Eradication** (removal) of pathogens from infested soil and tools and from infected seeds and infected propagative parts can be accomplished by various methods. **Protection** from disease involves the use of cultural practices, chemical and biological treatments, and genetic resistance. Not all methods of disease management are useful against all types of pathogens. The details of disease management and how specific methods are chosen are discussed in more detail later in this book.

Approaches to Plant Disease Management

Focused on the plant
- **protection**

Focused on the pathogen
- **avoidance**
- **exclusion**
- **eradication**

Focused on the environment
- **protection**

Plants exist in a world filled with potential pathogens. Many plant pathogens have adapted to their role in amazing ways. Plants have evolved equally interesting defense mechanisms. The interaction between plants and pathogens may result in disease, but, in fact, most plants are resistant to most diseases. To appreciate this dynamic interaction between plants and their pathogens, it is necessary to understand the basic biology of the major groups of pathogens. The next chapters introduce the biology of these groups and the diagnosis and management of the diseases they cause.

We can begin disease management plans by considering the disease triangle: the plant, the pathogen, and the environment. The disease cycle provides information about primary inoculum, infection courts, secondary inoculum, the

dispersal of primary and secondary inoculum, and the survival stage of the pathogen. Management options are commonly organized into four general categories: avoidance, exclusion, eradication, and protection.

REFERENCES

A basic resource, in addition to the works listed below, is the Plant Disease Compendium Series, published by The American Phytopathological Society (APS), St. Paul, MN, an ongoing series of more than 35 volumes, each covering a single crop or group of related crops, including fruit, vegetable, and field crops, turfgrasses, ornamentals, and flowering plants. Each volume contains many color illustrations, and several volumes have been translated into Spanish. A complete list of titles in this series is available at the APS website.

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Questions

1. What are the three components of the disease triangle? Create a disease triangle for apple scab and one for late blight of potato and tomato.
2. It is often recommended that a diagnosis should be made using plants or plant parts with a range of symptoms from early stages to late stages of disease—but not completely dead plants. Explain this recommendation.
3. What are Koch's four postulates for establishing proof of pathogenicity? Why is it not sufficient to simply isolate an organism from infected tissue and assert that it is the pathogen? Koch originally stated three postulates; the fourth was added later. Why do you think it was added?
4. Consider the general disease cycle, and list some advantages of growing annual plants as compared to perennial plants in terms of disease management.
5. If you were able to manage a disease with a fungicide, when should plants be protected if the disease is monocyclic? If it is polycyclic?

Challenge Questions

1. What are the important points of the life cycle of a new pathogen that need to be known in order to create an appropriate management program?
2. What are some advantages and disadvantages of the biotrophic and necrotrophic forms of parasitism? How would a disease caused by a biotroph differ from a disease caused by a necrotroph in terms of symptoms and continued survival of the host plant?
3. You have just joined the Peace Corps, and your assignment is to establish a plant disease diagnostic laboratory. Electricity will be unreliable. What basic equipment and materials will you request, and what kinds of diseases (e.g., pathogen identification) will you be able to diagnose?

Supplementary Resources

Illustrated Glossary

Special Topic

Plant disease diagnosis

Plant Disease Profiles

Monocyclic Diseases

Dutch elm disease
Stinking smut of wheat
Verticillium wilt

Polycyclic Diseases

Apple scab
Coffee rust
Late blight of potato and tomato
  Requeima (mildio, pt) da batateira e tomateiro (Portuguese)
  马铃薯和番茄晚疫病 (Chinese)

Case Study

Naughty peat: A case study in plant pathology, with emphasis on Koch’s postulates and disease etiology

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary of Plant Pathology. Additional words used in describing symptoms are listed in Exercise 1.1, with links to the Illustrated Glossary.

abiotic
avoidance
biotic
biotroph
colonize
direct penetration
disease
disease cycle
disease triangle
eradication
exclusion
facultative
facultative parasite
facultative saprophyte
fruiting body
germ theory

haustorium (pl. haustoria)
host range
incubation period
indirect penetration
infect
infection court
infectious
inoculate
Koch’s postulates
latent period
monocyclic
necrotic
necrotroph
noninfectious
obligate parasite
parasite
pathogen
pathogenicity
polycyclic
primary inoculum
protection
saprobe
saprophyte
saprotroph
secondary inoculum
sign
spontaneous generation
spore
symptom
transmit
vector

Exercise

Exercise 1.1. Identify the symptoms in the images presented.

Internet Resources

APS Resources

Effie A. Southworth, first woman plant pathologist hired at USDA
The future world food situation and the role of plant diseases
"On the famine trail"

Recommended Websites

APS Education Center
How To Methods: Making a Moist Chamber
Plants, Pathogens, and People

Internet Research

This exercise is designed to help you learn about specific diseases of plants of interest to you. Some economically important plants and a selection of their important diseases are listed in Appendix 2. You can find more extensive lists of host plants and their diseases in APS *Common Names of Plant Diseases*.

There are some excellent sites with images and fact sheets on specific plant diseases. Links to some of those sites are in the Internet Research section of the online resources.

Internet Research Exercise 1.1. Find information about a significant disease of a plant species that is of interest to you.

a. Make a plant disease triangle for this disease.

b. What kind of pathogen (fungus, bacterium, virus, nematode) causes the disease?
c. Is the pathogen a biotroph or a necrotroph?
d. Is the disease monocyclic or polycyclic?
e. Draw a disease cycle for this disease. Include the production and dispersal of primary inoculum, infection courts, the production and dispersal of secondary inoculum (if any), and the survival stage of the pathogen.

CHAPTER 2

What Are the Causes of Plant Diseases?

► Fungi

IN THIS CHAPTER:

- ► **What are fungi?**
- ► **What symptoms and signs do fungi cause?**
- ► **How do fungi survive and spread?**
- ► **How are fungal diseases diagnosed?**
- ► **How are fungal diseases managed?**
- ► **More detailed information about fungi, their reproduction, and fruiting bodies**

What are fungi?

Most plants and animals have a well-defined size and shape, but **fungi** are quite different. They are composed of living filaments that grow in a mass whose size and shape is determined by the environment. The filaments secrete extracellular enzymes to help degrade organic compounds and then absorb nutrients through their cell walls. All fungi are **heterotrophs**. That is, they require an outside food source and are incapable of producing their own food. Therefore, they seek nutrition from dead organic matter (as

saprophytes) or from living organisms (as parasites, either **necrotrophs** or **biotrophs**, of plants and animals). Apple scab (see Chapter 1), Dutch elm disease, and, in humans, athlete's foot are examples of diseases caused by fungi. Some parasitic fungi produce toxins (poisons) to aid in their invasion of the host, and some fungal compounds (mycotoxins) are toxic. Animals and humans who ingest poisonous mushrooms or plant materials contaminated by certain fungi can become ill or even die.

► DID YOU KNOW?

Fungal diseases of humans are not uncommon, especially in the hot, humid tropics. Ringworm, athlete's foot, and yeast infections are common diseases of animals caused by fungi that are generally restricted to the skin and nails. Other fungi, such as *Sporothrix schenckii*, which causes rose picker's disease, can infect below the skin only when introduced into a wound. Inhalation of certain spores can sometimes lead to lung infections. People with impaired immune systems, such as those with AIDS or diabetes, are susceptible to some systemic fungal diseases that are very difficult to control.

See Tom Volk's Fungi (website), Fungus of the Month, February 2003.

The growing filaments of a fungus form a web as they seek nutrients. An individual filament is a **hypha** (pl. hyphae), and the mass of hyphae that constitutes a fungus is the **mycelium**. (The term ***mycology***, referring to the study of fungi, is derived from the same root.) If a small piece of mycelium is placed on a food source, such as a nutrient medium in a petri dish, it will grow radially outward, forming a circle (Figure 2.1). Similar circles can be seen as ringworm (a fungal disease of the skin), circular areas of disease on tomatoes (Figure 2.2) and in turfgrass (Figure 2.3), round spots of mold on cheese and bread, and “fairy rings” of dark green grass or mushrooms in lawns and meadows. The shape of the mycelium can vary, depending on environmental conditions, such as nutrient availability. Hyphae may grow into mushrooms, puffballs, or other survival or spore-producing structures. Most of the characteristic hyphal and reproductive structures produced by fungi that cause plant diseases are microscopic.

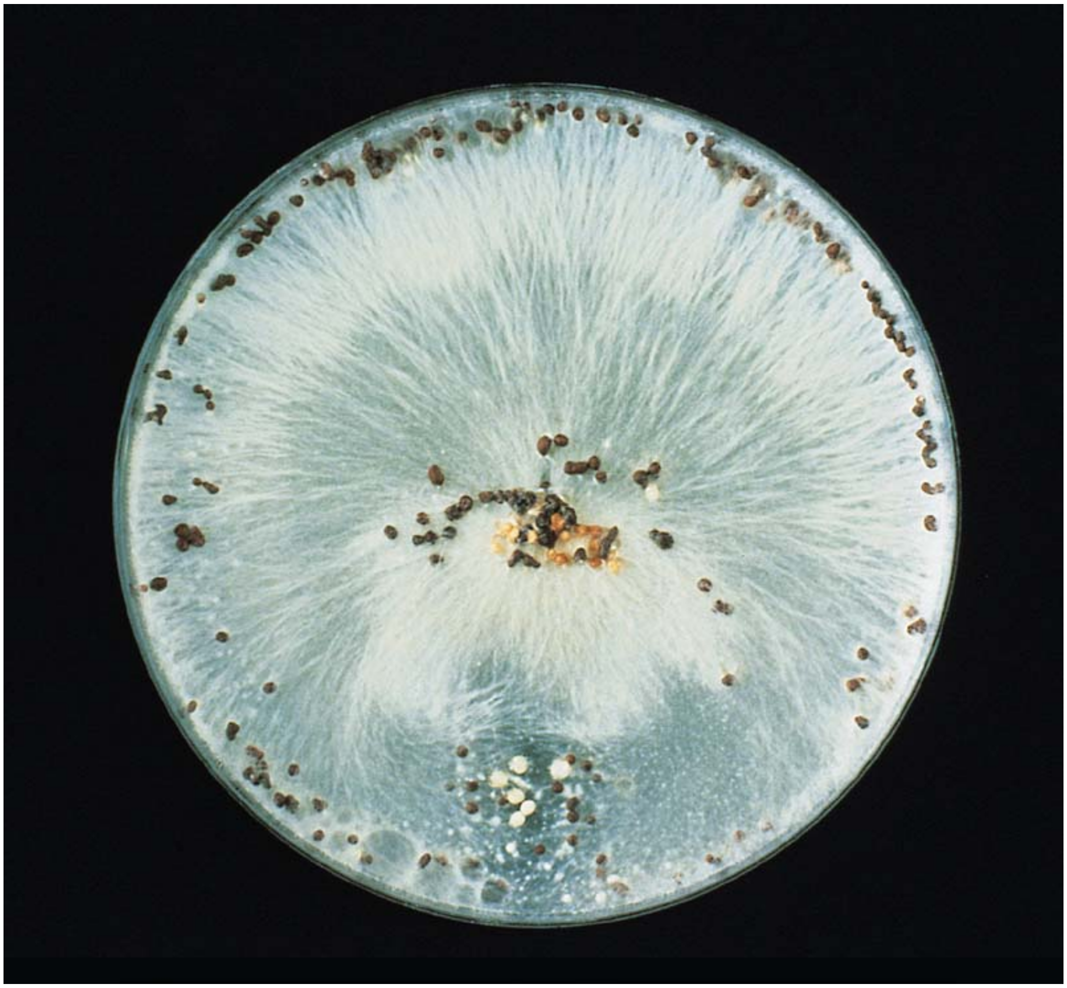


Figure 2.1. Mycelium and sclerotia (survival structures) of the fungus *Sclerotium rolfsii* on potato-dextrose agar.

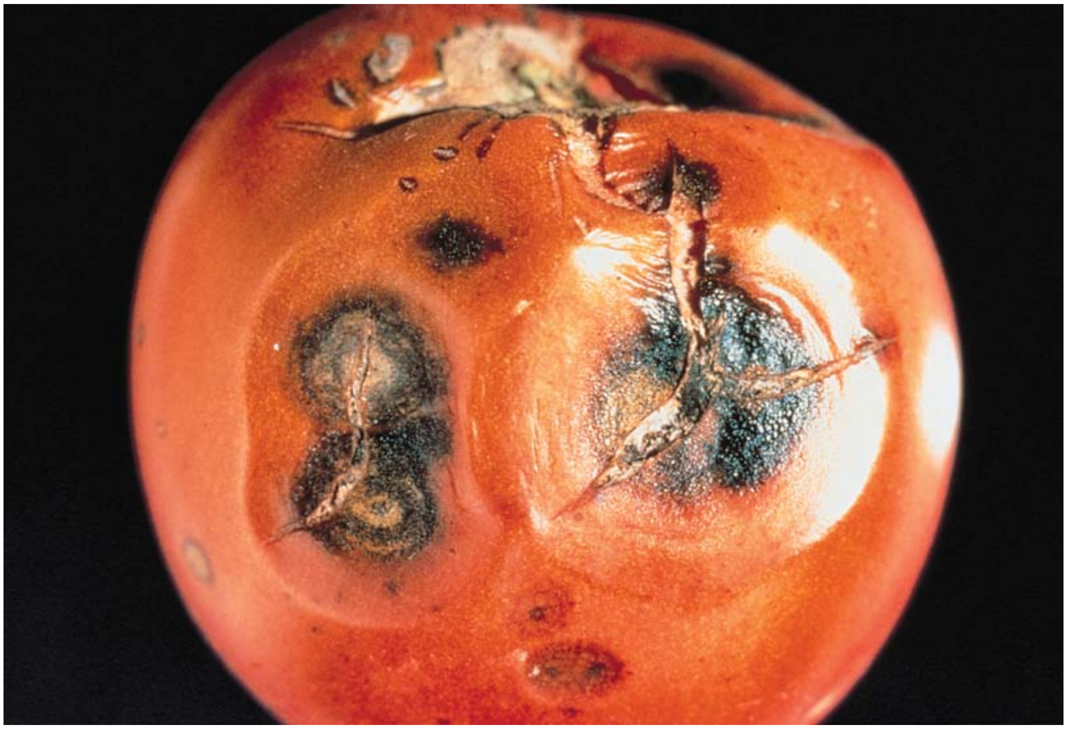


Figure 2.2. Tomato with anthracnose. The round spots of infection are typical of a fungal disease.

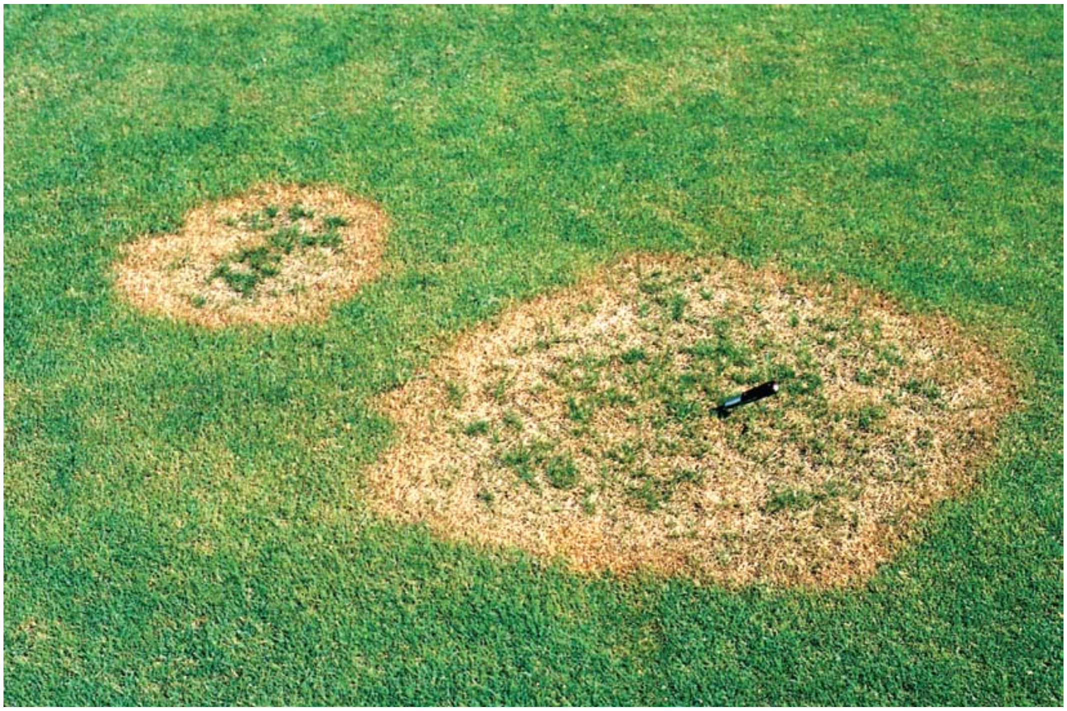


Figure 2.3. Take-all patch of bentgrass. The affected areas are circular, typical of fungal diseases.

As different as fungi may seem to be from animals and plants, they are very similar at the cellular level. Cells of hyphae contain mitochondria, nuclei, and other organelles, just like the cells of all **eukaryotic** organisms. Nuclei in fungal hyphae are **haploid** (1N), having only one copy of each type of chromosome, in contrast to the nuclei of most eukaryotes, in which the diploid phase is dominant. A cell wall surrounds the cytoplasm of hyphae. Unlike the cellulose-rich cell wall of plants, fungal cell walls are predominantly **chitin**, the same compound found in the shells of crabs and lobsters and the exoskeletons of insects. Fungi use glycogen for food storage, as animals do, and not starch, as plants do. Such similarities, together with DNA and RNA sequences, suggest that fungi are more closely related to animals than to plants. Older textbooks have described fungi as plants without chlorophyll, but this is a legacy from the early days of biology, when scientists attempted to classify all organisms as either plants or animals. Because fungi are not mobile and have cell walls, they were regarded as a subject for study by botanists. Much of the terminology used to describe fungi is similar to botanical terminology because of this history.

Fungi are composed of living filaments called hyphae, which form a mycelium. The cells of hyphae have nuclei, mitochondria, and other organelles. The cell walls contain chitin.

Characteristics of True Fungi

- **composed of** hyphae, which form mycelia
- **cell wall**: predominantly chitin with glucan (a complex sugar)
- **storage compound**: glycogen

- **cell structure**: eukaryotic (with haploid nuclei)
- **nutrition**: heterotrophic; nutrients absorbed through cell walls
- **reproduction**: spores (sexual or asexual)
- **nonmotile**

In many fungi, the cells of the hyphae have cross-walls, called septa. A **septum** is a divider, such as the septum that separates the nose into two nostrils. Hyphae with cross-walls are said to be **septate** (Figure 2.4A). In some fungi, however, the hyphae are **nonseptate** (**aseptate** or **coenocytic**) (Figure 2.4B) and contain multiple nuclei. Other fungi have a variable number of nuclei between septa, and still others have a pair of nuclei between septa. The structure of a fungal septum varies, depending on the fungal group. In some fungi, cellular material, including nuclei, can pass through a pore in the septum. Thus, it is not always possible to identify a single "cell" of a fungus. It also is not possible to see fungal nuclei without special stains. (Novices often confuse oil droplets in the cytoplasm with nuclei when hyphae are viewed with a microscope.)

For diagnostic purposes, the presence or absence of septa in the hyphae is an important feature that helps determine to which of the major fungal groups an organism belongs (see the table on this page). In some fungi, a hump-shaped structure, a **clamp connection** (Figure 2.5), is present on some septa; this characteristic identifies these fungi as members of one specific group, the basidiomycetes.

True Fungal Group	Hyphal Characteristics

ascomycetes	septate hyphae
basidiomycetes	septate hyphae; some but not all species have clamp connections
chytridiomycetes	lack true mycelium; thallus often irregularly shaped
zygomycetes	nonseptate (coenocytic, aseptate) hyphae

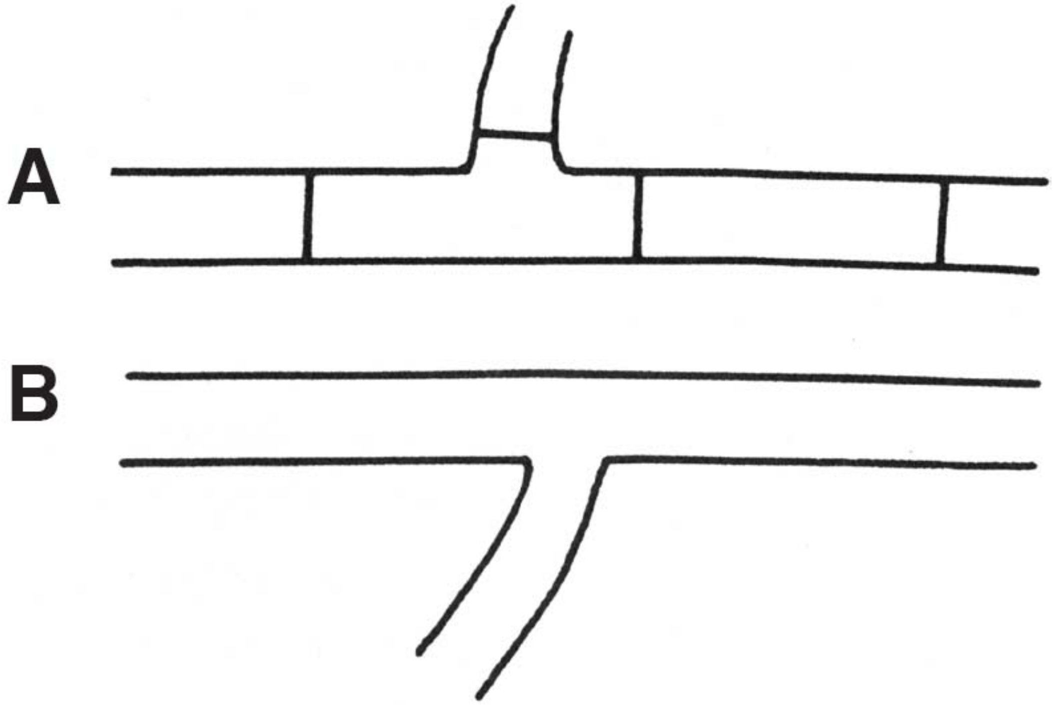


Figure 2.4. Septate (A) and nonseptate hyphae (B). Note the cross-walls in the septate hypha.

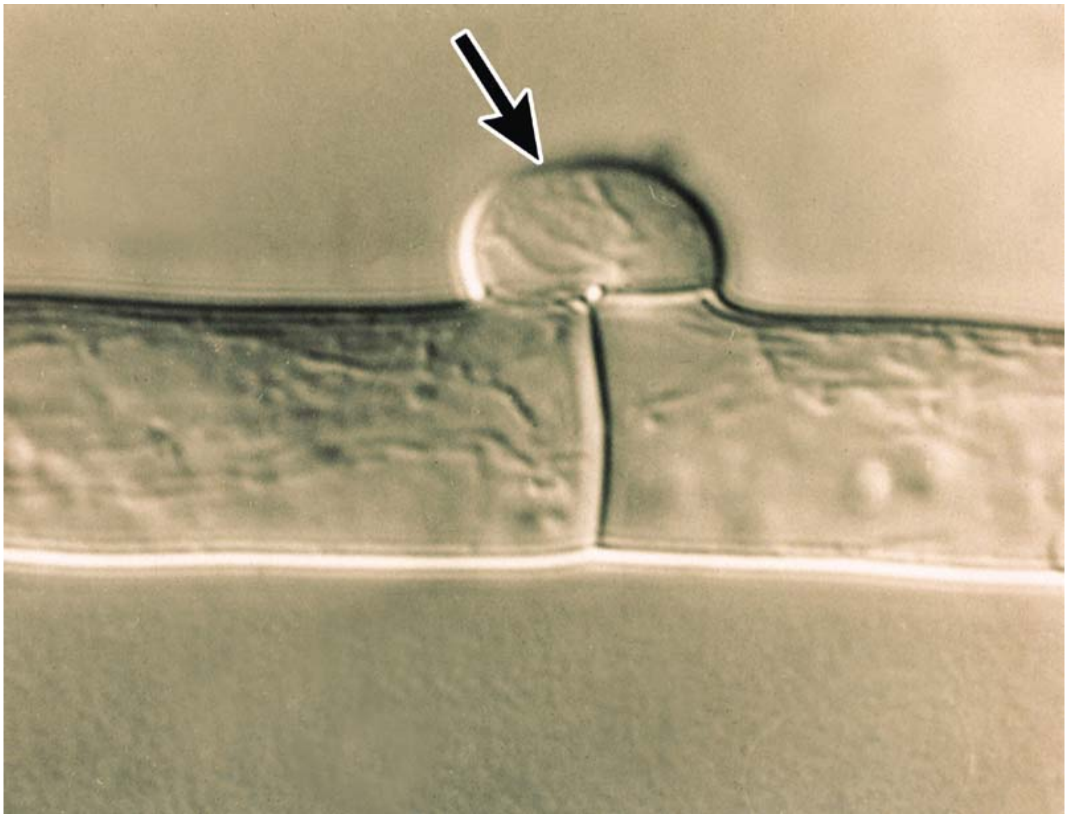


Figure 2.5. Hypha with a septum and a clamp connection (arrow).

Fungi reproduce by means of **spores** (Figure 2.6). In early studies of fungi, the spores were called seeds, by analogy with the seeds of plants. Spores are important for the survival and dispersal of fungi. Most hyphae are fragile and cannot withstand environmental extremes, particularly desiccation (drying out).

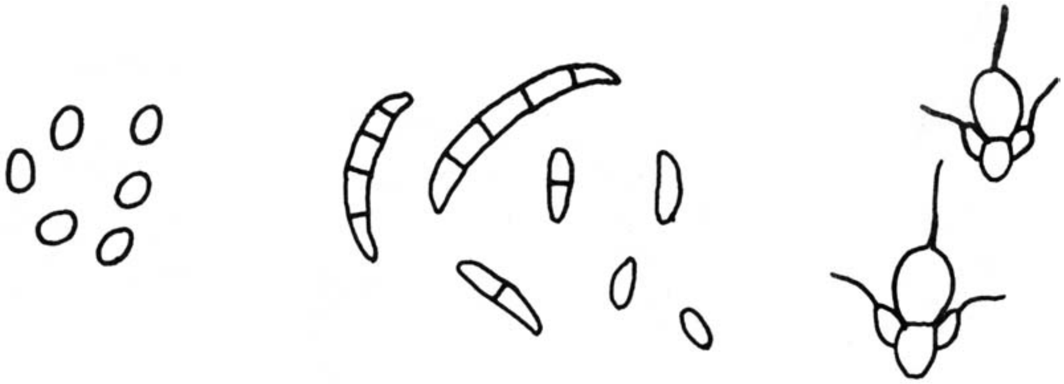


Figure 2.6. Fungal spores of various shapes.

Most fungi produce at least two kinds of spores: sexual and asexual. **Sexual spores** are produced in the same sexual cycle that occurs in all eukaryotic organisms. The key events in the sexual cycle (Figure 2.7) are

- **plasmogamy**: fusion of cells, bringing two sexually compatible haploid nuclei together in one cell
- **karyogamy**: nuclear fusion, forming a diploid nucleus
- **meiosis**: formation of four genetically recombined haploid nuclei from a single diploid nucleus

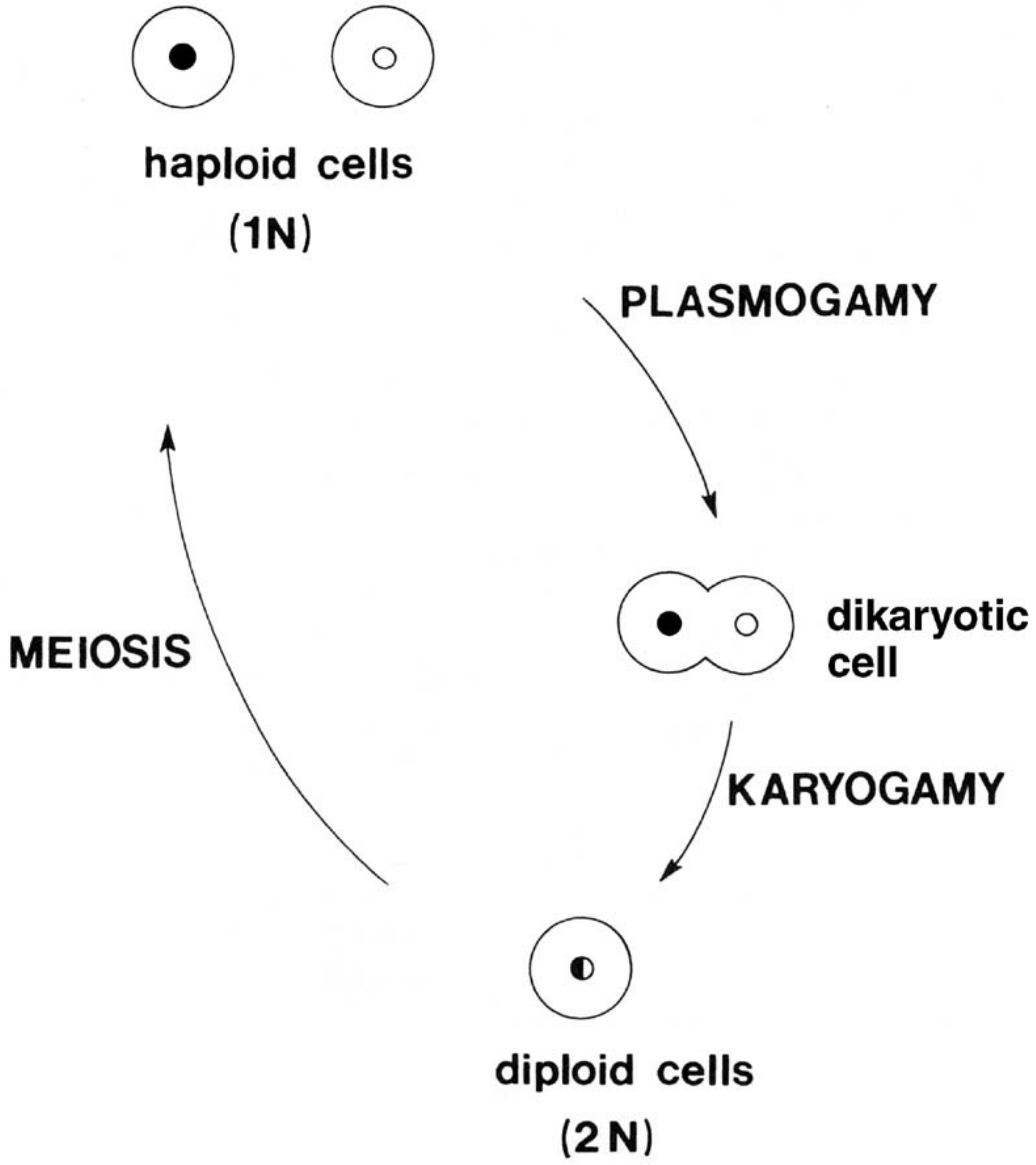

haploid cells
(1N)
PLASMOGAMY
dikaryotic cell
KARYOGAMY
diploid cells
(2N)
MEIOSIS

Figure 2.7. The sexual cycle.

teleomorph
- perfect stage
- sexual spores only

anamorph
- imperfect stage
- asexual spores only

holomorph
- spores in all stages, sexual and asexual

The purpose of sex in fungi is the same as in other organisms: to provide genetic diversity for the continued ability of the species to adapt to changing conditions. This adaptability has enabled some fungi to overcome plant resistance genes selected by plant breeders to protect plants from infection, and it has enabled some fungi to become resistant to certain fungicides.

Commonly, the sexual spores are the survival spores of the fungal life cycle. They may help the fungus overwinter or, especially in arid regions, oversummer. Hyphae grow into special aggregations called **fruiting bodies**, which produce new spores. Spores are usually produced on the surface of a fruiting body or inside it, where they are protected. Desiccation is an important cause of spore death, so fruiting structures are often composed of cells with thickened walls that help prevent the loss of moisture during dry periods, including winter when the air is dry. Also, the cell walls of fruiting bodies may contain **melanin**, a dark pigment that helps protect against ultraviolet radiation from the sun.

Most fungi also produce at least one kind of **asexual spore**. These spores are genetically the same as the parent mycelium, since there is no change in nuclear condition during asexual reproduction. Asexual spores are essentially "packets" (clones) of the fungus, containing at least one nucleus and enough cellular material to allow a new mycelium to grow when the spore germinates. Some spores are powdery and adapted for air dispersal; other spores are produced in a sticky mass that is dispersed by splashing raindrops, agricultural tools and machinery, or a passing insect or other animal. Many fungi produce a type of asexual spore called a **conidium** (the name is derived from the Greek word for dust). The dusty spores observed on moldy bread and cheese are **conidia**.

Spores are the reproductive units of fungi. Most fungi produce sexual spores, which provide genetic variation. Most fungi also produce one or more kinds of asexual spores, which aid in dispersal.

► DID YOU KNOW?

Armillaria gallica achieved fame in 1992 when its mycelium was found across 37 acres of forest in Crystal Falls, Michigan. It was calculated that the mycelium in the soil weighed more than 110 tons, about the same as an adult blue whale, and originated at least 1,500 years ago. This basidiomycete moves from tree to tree by growing dark, cordlike rhizomorphs, giving it its common name, the shoestring fungus. The fungus produces clumps of mushrooms at the bases of infected trees. Since then, *Armillaria ostoyae* has been found covering 2,200 acres in Oregon and may be 2,400 years old. Claims that this fungus was one of the oldest and largest living organisms were later challenged by scientists studying clonal aspens in the western United States. But the discovery of the "humongous fungus" started an annual festival in Crystal Falls and was even featured on the side of some U-Haul trailers.

See Tom Volk's Fungi (website), Fungus of the Month, April 2002.

What symptoms and signs do fungi cause?

Fungi cause a wide variety of plant diseases. The symptoms they cause vary depending on the type of plant and the part of the plant that is infected. Because fungi tend to grow out radially from a central starting point, the classic leaf spot symptom of a fungal pathogen is circular. These **circular spots** may be blocked by large leaf veins or elongated into ovals between the parallel veins of monocot leaves, such as those of corn or iris. Canker fungi invade the phloem and vascular cambium, causing sunken areas on stems and twigs. Vascular wilt fungi invade the xylem, impeding water transport and thus leading to **wilt** and **discoloration of the active xylem**. Plants with **root rots** are often stunted and wilted and have symptoms of nutrient deficiency, and their roots may be brown and deteriorated.

Many of these symptoms can also be caused by other kinds of pathogens, so it is helpful to examine diseased plants for **signs** of specific fungal pathogens. **Mycelia, spores** (Figure 2.8), and **fruiting bodies** (Figure 2.9) may be observed. If no fungal signs are obvious, incubate a sample of infected tissue overnight in a moist chamber (such as a closed container with moist paper towels in it). This may stimulate the fungus to grow out onto the surface of the tissue and produce mycelia and spores, which can be examined microscopically to identify the pathogen. Differentiating between a pathogen and a harmless saprophyte can be challenging, even for an experienced diagnostician.

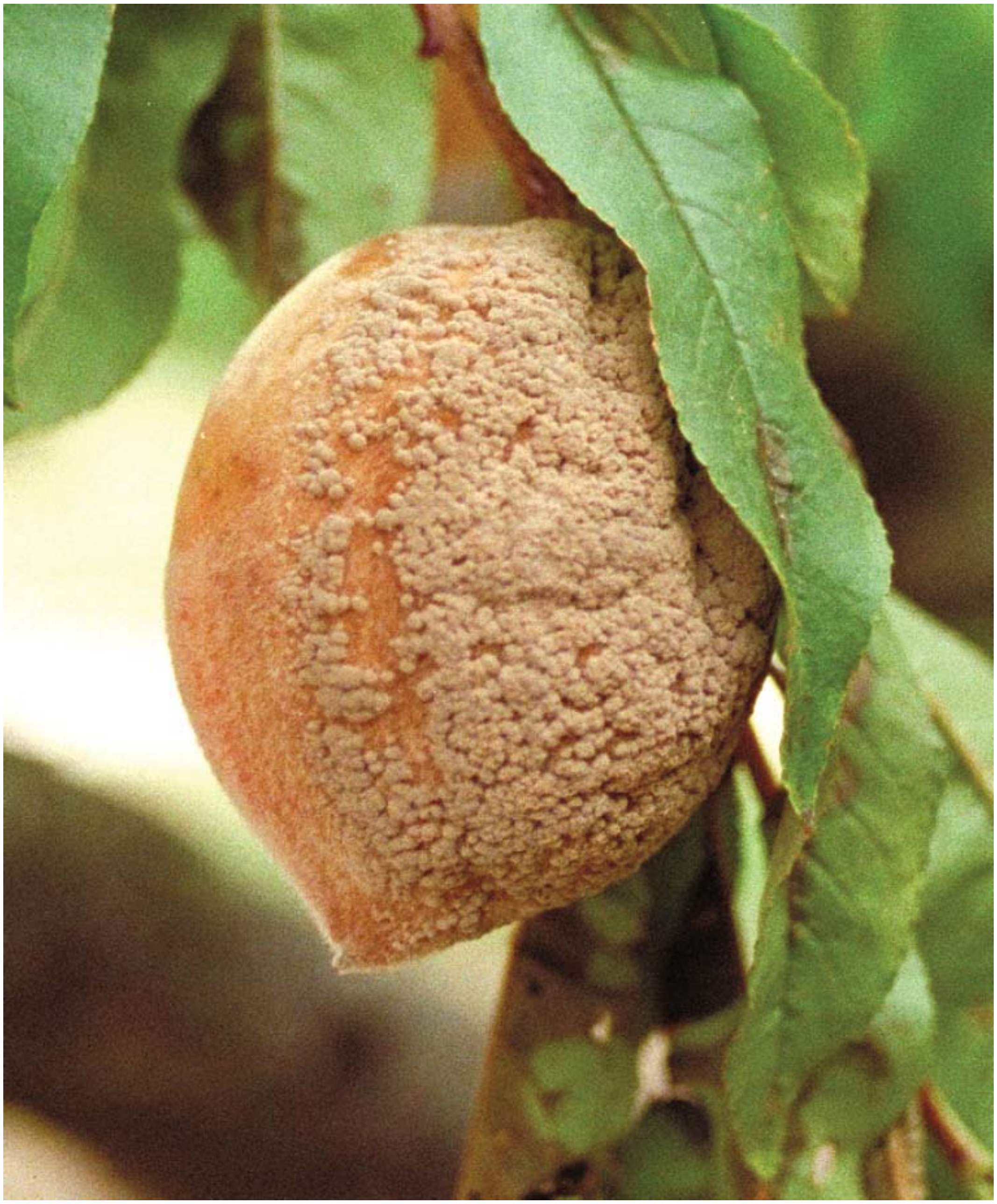


Figure 2.8. Peach with brown rot. The affected area is covered with spores, a sign of the pathogen.

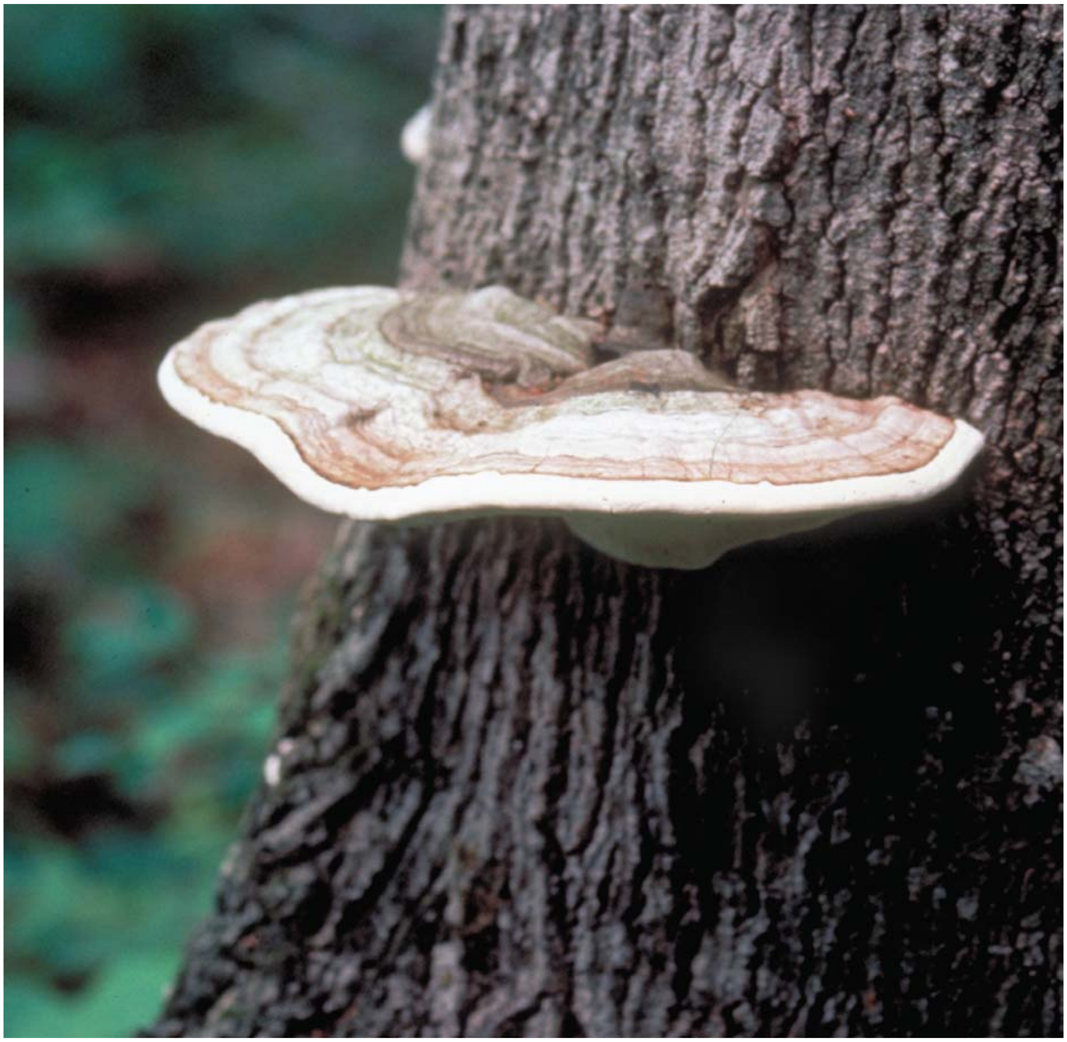


Figure 2.9. Fruiting body (conk) of a wood decay fungus, a sign of the pathogen.

In descriptions of fungi that cause plant diseases, a pathogen may be referred to by two or more scientific (Latin) names: one name refers to the **teleomorph** (the sexual stage of the fungus), and one or more names refer to the **anamorph** (the asexual stage or stages). The name of the teleomorph applies to the whole organism in all its stages (also known as the **holomorph**). Separate names for anamorphs are still commonly used, because the asexual stage is often produced soon after infection and may be the only stage observed until the end of the season or even the beginning of the next season. To diagnose a fungal disease as soon as possible, it may be necessary to identify the pathogen initially by the characteristics of its anamorph. Once the organism is identified, the holomorph name is sometimes used, even though the sexual stage may not have been seen.

The scientific names of organisms are Latin binomials (two names): a **genus** name (capitalized) and a **species** name, or **specific epithet** (not capitalized). Scientific names should be underlined (or italicized when possible).
Example *Armillaria gallica*

After the entire species name has been mentioned in a text, the genus can then be abbreviated to its first letter if the name is used again.
Example *A. gallica*

When the genus name is followed by **sp.**, the organism has not been identified to species.
Example *Armillaria* sp.

A genus name followed by **spp.** is a plural form that refers to more than one species belonging to that genus.
Example *Armillaria* spp.

A genus name that is used in the name of a disease is capitalized but not italicized.
Example Armillaria root disease

The sexual stage of some fungi is unknown (or may not even exist), so the anamorph name is the only name. These fungi were formerly called **imperfect fungi** (or Fungi Imperfecti or **deuteromycetes**), because either they lack the perfect (sexual) stage or it has not yet been found. Most of these asexual fungi are ascomycetes, but some are basidiomycetes with asexual states (see below). To avoid confusion, remember that the mycelium is the organism and that the different spore types are simply the reproductive stages that allow the fungus to survive and disperse and that help us to recognize and identify the fungus.

► **DID YOU KNOW?**

About 300,000 species of fungi have been described, but this number may be only about 20% of the fungal species on earth, because so many regions remain to be studied in detail, especially in the tropics. In the book *Fungi on Plants and Plant Products in the United States*, 78,000 unique fungus-host combinations are listed, with 13,000 accepted fungal names.

It is impossible to identify most fungi by observing only the characteristics of their mycelia. It is usually necessary to observe the spores or the fruiting bodies (or both). It can be overwhelming for beginners to look at the long list of specialized terms and taxonomic names associated with fungi. However, there are four major groups of true fungi, and each has some characteristic spores and fruiting bodies. Once you are familiar with the words describing these groups, you will find many plant pathology resources easier to understand and to use for disease diagnosis.

All parts of a plant are subject to fungal diseases. When leaves are infected by fungi, the spots are often circular. Diagnosis requires observation of the spores and fruiting bodies so that the pathogen can be positively identified. The

anamorph (asexual stage) may be the only stage present, especially in the early stages of a fungal disease.

Until recently, most evidence of the phylogeny (evolutionary development and history) of fungi has been based on morphology (appearance), development, genetics, and physiology. In molecular systematics studies, comparisons of genetic sequences are used to improve our understanding of biological relationships. The molecular studies have confirmed, for example, that the Oomycota and some other groups are not true fungi. They also confirmed that the true fungi comprise a group of organisms that share a common ancestor. The new evidence also is helping to determine appropriate taxonomy at the lower levels, such as orders, families, and genera. The website Deep Hypha, sponsored by the National Science Foundation, was organized to improve communication among scientists who study the phylogenetics of fungi using both traditional and molecular methods.

Plant Pathogens Classified as True Fungi

The four major groups (phyla) of true fungi are Ascomycota, Basidiomycota, Chytridiomycota, and Zygomycota. The first two groups contain many important plant pathogens, whereas the last two groups contain very few.

There are some other fungus-like organisms, such as the Oomycota, that cause significant plant diseases but are now known to belong to different evolutionary lineages. Although they no longer are classified in the Kingdom Fungi, they are more similar to fungi, with respect to the kinds of plant diseases they cause and how we manage them, than to other major pathogen groups such as bacteria, nematodes, or viruses. Many of them have adaptations for dispersal, infection, and survival similar to those of true fungi despite their disparate origins. Short discussions of these fungus-like organisms follow this section.

ASCOMYCOTA

ASCOMYCETES

- **septate** hyphae
- sexual spores: **ascospores**, formed in an **ascus** (sac)
- asexual spores: **conidia**

The fungi in this group are commonly referred to as ascomycetes. They include bread and beer yeasts, morel mushrooms, and plant parasites that cause many well-known diseases, such as apple scab, chestnut blight, and Dutch elm disease, as well as cankers, leaf spots, postharvest diseases, powdery mildews, and vascular wilts.

Ascomycetes are characterized by sexual spores, **ascospores**, which are produced in an **ascus** (pl. asci) or sac (Figure 2.10). Sometimes the asci are exposed on the surface of a leaf or other plant tissue, but usually they are formed inside a fruiting body. The general term for a fruiting body that contains asci and ascospores is **ascoma** (pl. ascomata) or **ascocarp**. The fruiting bodies of most plant pathogens are just barely visible or are microscopic. There are several types of ascomata. Some simply crack open to release the spores, but most have an opening (**ostiole**) or open surface for spore release. The details of reproduction and the specific fruiting bodies produced by ascomycetes and some other fungal groups are described in more detail at the end of this chapter.

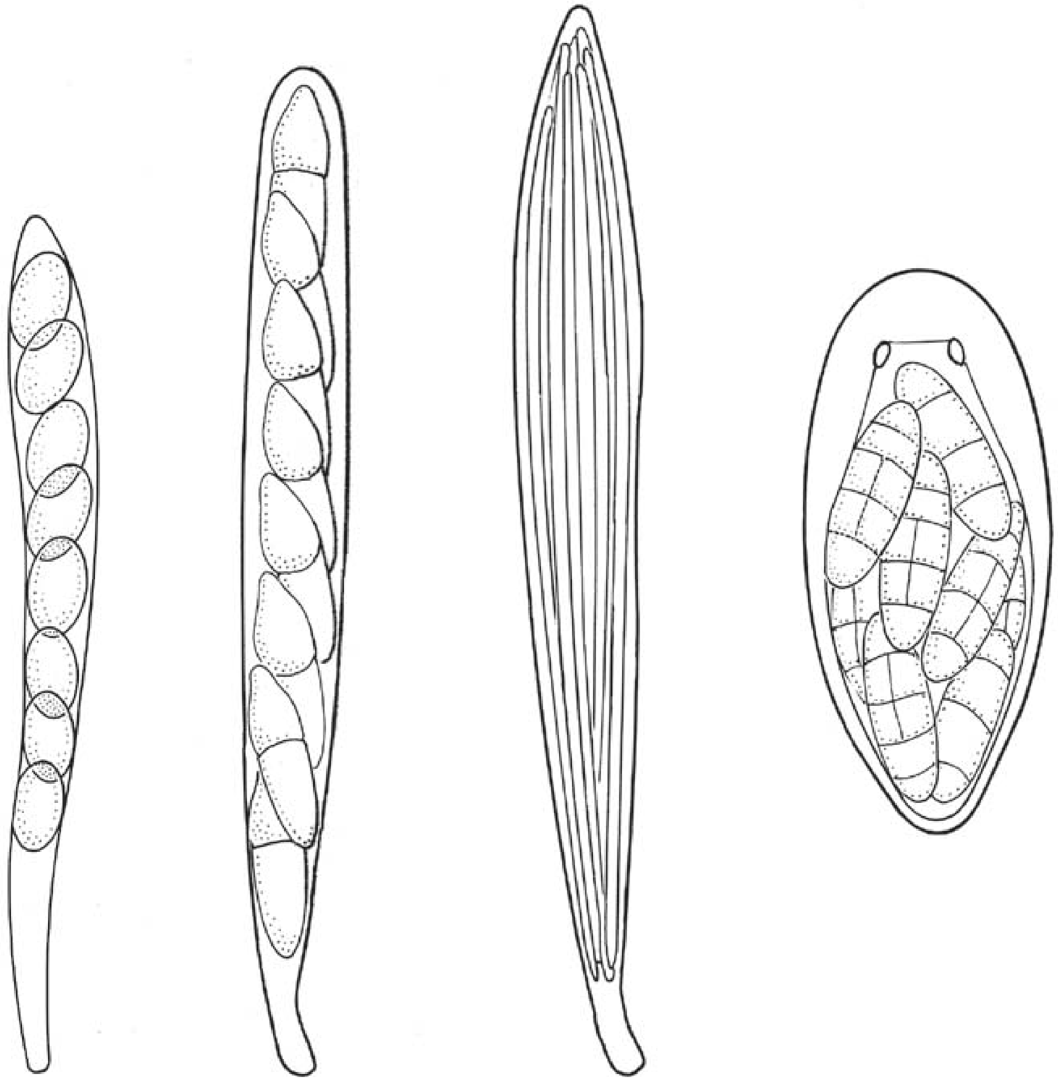


Figure 2.10. Examples of asci (the outer sac in each diagram) and ascospores (usually eight per ascus). The shape and size of the ascus and ascospores vary greatly.

Conidia are asexual spores commonly produced by the anamorphs (asexual stages) of ascomycetes. Ascomycetes that produce only asexual spores were formerly classified as deuteromycetes (imperfect fungi). Conidia and **conidiophores**, the specialized hyphae that produce conidia, may be produced on the surface of a host plant (or other substrate) or in a fruiting body. The general term for an asexual fruiting body is **conidioma** (pl. conidiomata), and there are several types.

To determine whether a disease is caused by an ascomycete, it is helpful to observe the hyphae, which are septate, and to find spores and/or fruiting bodies. In the absence of a fruiting body or supporting hyphae, it is not possible to tell whether an individual spore is sexual or asexual by microscopic examination. Spores produced in asci are ascospores and, therefore, sexual. When asci and ascospores are observed, then the fungus is certainly an ascomycete because these define the fungal group. Identification to genus or species is traditionally based on the color, size, and shape of the spores (ascospores or conidia) and the type of fruiting body (ascoma or conidioma) in or on which they are found.

DISEASE CLASSIC

Ergot of Cereals and Grasses

Hosts rye and other cereals; some cool-season grasses

Pathogen *Claviceps purpurea* (an ascomycete)

Symptoms and Signs Only the ovary of the host is infected. The seed or grain is replaced by the survival structure (sclerotium) of the fungus, called an ergot.

Disease Cycle Tiny fruiting bodies sprout from ergots on the soil surface at the time of host flowering. Ascospores (primary inoculum) are forcibly discharged from the fruiting bodies and may be trapped by the feathery surface of the stigma of cereal or grass flowers. Ascospores germinate there and infect the ovary. A **honeydew** containing conidia (secondary inoculum) is produced on the surface of infected ovaries. Insects and water aid in the dispersal of conidia, which initiate new infections.

Management Deep plowing to bury ergots prevents germination of sclerotia and, therefore, the production of ascospores. Crop rotation to a nonsusceptible crop is recommended, especially where minimal tillage is practiced. Mowing of wild grasses reduces inoculum. If ergot-contaminated seed is used, ergots should be removed or seeds should be planted deeply to prevent ascospore production.

Significance The ergot fungus produces numerous alkaloids that cause serious health effects in humans who ingest flour contaminated with ergots. Animals fed contaminated grain may also become ill. Ergotism was known as St. Anthony's Fire or the Holy Fire in the Middle Ages, before people made the connection between the ingestion of ergots and miscarriage, gangrene in the hands and feet, and hallucinations.

Plant Disease Profiles: Ergot

Important Ascomycetes

Blumeria graminis (powdery mildew of cereals and turf)

Botrytis spp. (gray mold)

Diplocarpon rosae (black spot of rose)

Fusarium oxysporum (Fusarium wilt)

Gibberella zeae (wheat scab)

Magnaporthe grisea (rice blast)

Monilinia fructicola (brown rot of stone fruits)

Mycosphaerella fijiensis (black Sigatoka of banana)

Ophiostoma ulmi (Dutch elm disease)

Sclerotinia homoeocarpa (dollar spot of turf)

Venturia inaequalis (apple scab)

► **DID YOU KNOW?**

The ascomycete *Monosporascus cannonballus* is named for its unusual ascospore. Asci typically contain eight ascospores, but *M. cannonballus* produces a single, large, dark ascospore: a cannonball.

Plant Disease Profiles: Monosporascus root rot and vine decline of melons

Ascomycetes produce sexual ascospores in asci. Each species has a single characteristic type of ascospore and also may produce one or more kinds of conidia (or other asexual spores). An ascoma or ascocarp (sexual fruiting body) and/or a conidioma (asexual fruiting body) may be present. The hyphae are septate. Ascomycetes cause a wide range of plant diseases and include species that range from obligate parasites to facultative parasites and saprophytes to obligate saprophytes.

DISEASE CLASSIC

Powdery Mildew of Cereals and Grasses

Hosts wheat, barley, cool-season grasses (e.g., Kentucky bluegrass)

Pathogen *Blumeria graminis*, syn. *Erysiphe graminis* (an ascomycete). This species is distinguished into various host-specific forms (formae speciales); *B. graminis* f. sp. *tritici* infects wheat.

Symptoms and Signs Powdery mildews are easily recognized by their white mycelium and conidia, mostly on the upper surfaces of leaves. Later, sexual fruiting bodies called chasmothecia (cleistothecia) form. The chasmothecia are dark, closed bodies that look like tiny, dark basketballs.

Disease Cycle The chasmothecia rupture to release ascospores (primary inoculum). The mycelium of most powdery mildew fungi remains on the surface, absorbing nutrients via haustoria in the epidermal cells of leaves and fruit. White, oval conidia (secondary inoculum) are produced in chains from the mycelium, initiating secondary infections.

Management In Europe, fungicides are routinely used for powdery mildew of cereals. Many U.S. cereal crops grown in less humid climates and turfgrasses where the leaves are mowed do not require fungicides. Planting cultivars with genetic resistance is a common practice. Rotation to a nonhost crop and removal of infected volunteer plants are recommended.

BASIDIOMYCOTA

BASIDIOMYCETES

- **septate** hyphae; **some** species have **clamp connections** at some septa
- sexual spores: **basidiospores**, generally produced in a group of four external to the **basidium** (the cell that produces them)
- asexual spores: vary considerably within this group

The fungi in this group are commonly called basidiomycetes. They include most of the fungi that people are familiar with from fields and forests: mushrooms, puffballs, bird’s nest fungi, and conks

(Figure 2.9) and other fruiting bodies found on dead and dying trees. Most of the wood decay pathogens are in this group, but not all basidiomycetes found in association with trees are harmful. Many mushroom-producing fungi are beneficial to trees and other kinds of plants because they are ectomycorrhizal fungi. Hyphae of these fungi form a protective web of mycelium on host roots and aid in nutrient and water absorption in exchange for nourishment from the tree. In addition to the species that produce large fruiting bodies, there are some important microscopic basidiomycetes that cause significant plant diseases, including *Rhizoctonia* species and *Sclerotium rolfsii*. Plant-pathogenic **rust fungi** and **smut fungi**, although quite different from mushroom-producing fungi, are included in this group because they share features of sexual reproduction and are part of the same evolutionary heritage.

Many, but not all, basidiomycetes have **clamp connections** (Figure 2.5) on their hyphae at some of their septa. No other fungal group has this structure. Most basidiomycetes are **dikaryotic** for most of their lives, which means that each cell of the mycelium contains a pair of sexually compatible haploid nuclei that divide at the same time during mycelial growth. As a result of sexual reproduction, four haploid nuclei are usually formed in a cell called the **basidium** (pl. basidia) (Figure 2.11). These nuclei migrate into four externally produced **basidiospores**, one nucleus per spore. The gills and pores of mushrooms and other fruiting bodies are lined with numerous basidia, each of which usually produces four basidiospores. A sexual fruiting body of a basidiomycete is called a **basidioma** (pl. basidiomata) or a **basidiocarp**. Different terminology is used for fruiting bodies of rust and smut fungi.

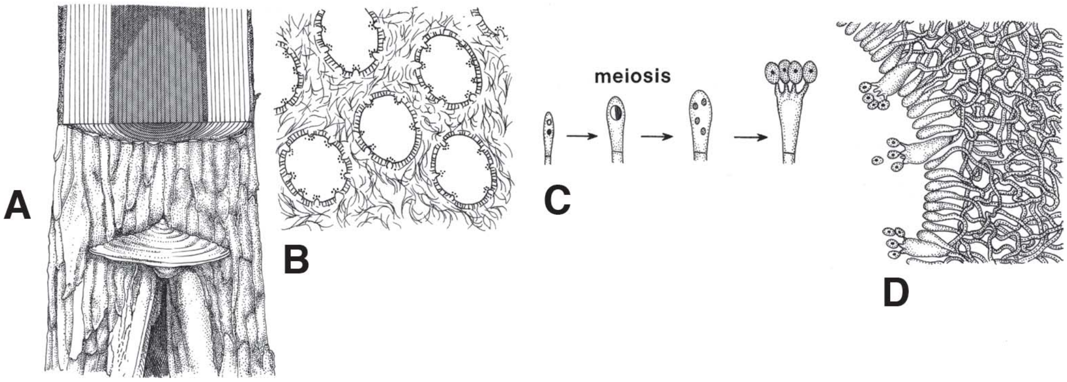


Figure 2.11. Reproduction of a basidiomycete. A, Fruiting body. B, The lower surface of a fruiting body, containing numerous pores. C, Formation of basidiospores following meiosis. D, Basidia and basidiospores lining the pores in a fruiting body.

► DID YOU KNOW?

Basidiospores can be observed by placing a mushroom cap, with the gills down, on a piece of paper and covering it with a bowl for a few hours. Spore color cannot be determined by looking at the gills, so it is best to place one mushroom cap on a light-colored piece of paper and one on a dark-colored piece of paper. This provides contrast for observing the released spores.

In most cases, the haploid basidiospores germinate to form a haploid mycelium, which, in various ways depending on the species, must fuse with a compatible haploid mycelium or spore to reestablish the dikaryotic state.

Smut fungi (Figure 2.12) are named for the massive numbers of dustlike, dark, thick-walled **teliospores** that they produce. The word *smut* comes from a Germanic word for dirt. Germinating teliospores produce a haploid stage that fuses with that of another germinating teliospore to reestablish the dikaryotic mycelium necessary for infection.

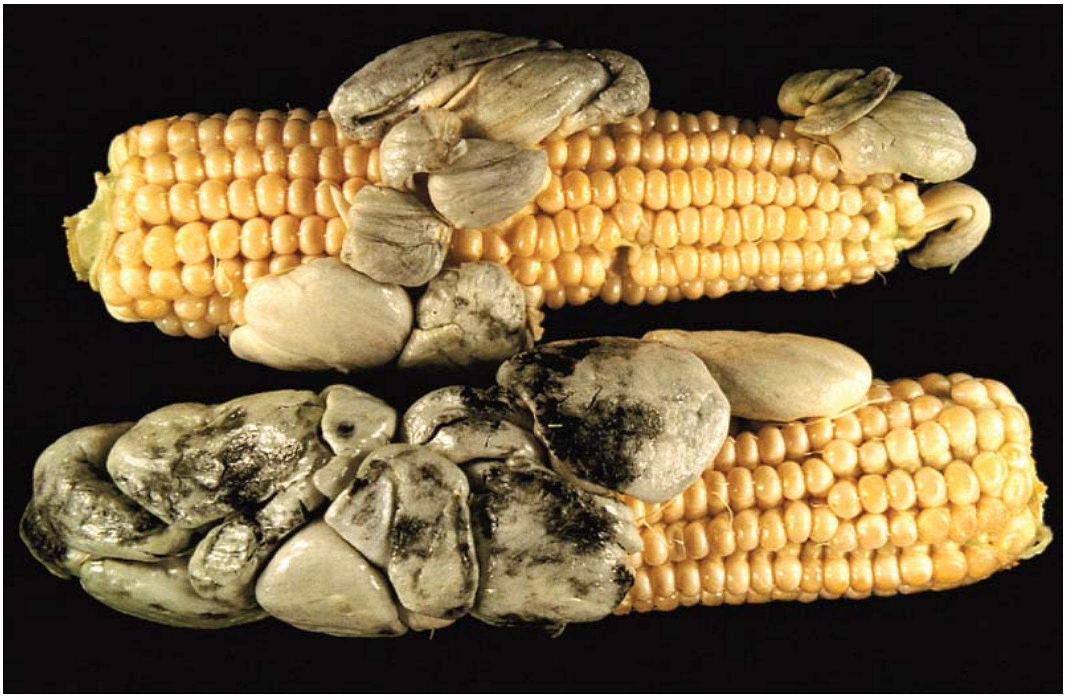


Figure 2.12. Corn smut, caused by *Ustilago maydis*, a basidiomycete.

► **DID YOU KNOW?**

Corn infected with smut, caused by the basidiomycete *Ustilago maydis*, has long been cultivated and sold in Mexico. It is even available in cans, as mushrooms are in supermarkets in other countries. It is known as *cuitlacoche*, derived from an Indian word for dung. Renamed the smoky maize mushroom for gourmet markets and specialty restaurants in the United States, smut-infected corn is cultivated in several states, because it can be sold for a much higher price than uninfected corn.

Rust fungi have complex life cycles that can include up to five spore stages and two unrelated host plants. Like smut fungi, they produce dark teliospores from which basidiospores are produced. Rust diseases are named for the asexual **urediniospores**, which are often orange. The powdery urediniospores are produced in numerous blisterlike pustules and can cause repeated infections of the host plant and potentially massive epidemics. Rust fungi are biotrophs (obligate parasites) and cause foliage and stem diseases of a wide variety of plants, but each rust species typically can infect only its specific host plants.

Important Basidiomycetes

Rhizoctonia spp. (root rots and turfgrass diseases)

Rust fungi

Sclerotium rolfsii (southern blight)

Smut fungi

Wood decay fungi

DISEASE CLASSIC

Armillaria Root Disease

Hosts stressed hardwood trees in the eastern United States; conifers in the western United States

Pathogens *Armillaria mellea* and other *Armillaria* species (basidiomycetes)

Symptoms and Signs Roots become decayed, resulting in an overall decline of the infected tree above ground. White mycelial fans form under the bark at the base of the tree. Clumps of honey-colored mushrooms occasionally form at the base of the tree. Black, shoestringlike rhizomorphs are common on the surface of infected roots.

Disease Cycle *Armillaria* spp. are root-rotting fungi that move from tree to tree in the soil via black rhizomorphs. Clumps of mushrooms form at the bases of infected trees. They are the source of airborne basidiospores. The hyphae of germinating basidiospores must fuse to reestablish a dikaryotic mycelium capable of infecting host trees.

Significance These important root pathogens can cause so much damage that infected trees may fall over during windstorms. The mycelium of *Armillaria* is known for its bio-luminescence, a greenish glow known as foxfire. Infected pieces of wood can be used to light one's way on moonless nights. This is the "humongous fungus" described earlier in this chapter (p. 24).

Plant Disease Profiles: Armillaria root disease

Basidiomycetes produce sexual basidiospores and sometimes other types of spores. The hyphae are septate and may have a clamp connection at some or all septa, depending on the species. This diverse group includes most mushrooms, conks, rust fungi, and smut fungi. Like ascomycetes, basidiomycetes cause a wide range of plant diseases and include species that range from obligate parasites to facultative parasites and saprophytes to obligate saprophytes.

CHYTRIDIOMYCOTA

Most species in this group of organisms are saprophytes, but the plant-parasitic members are all biotrophs (obligate parasites), so they cannot be grown in culture plates. Chytridiomycetes, often called chytrids, have a limited mycelium that is restricted to the cells of the host. Three plant-pathogenic genera are commonly noted: *Physoderma* (e.g., brown spot of corn), *Synchytrium* (e.g., wart of potato), and *Urophlyctis* (e.g., crown wart of alfalfa). Unlike most fungi, members of this group produce **zoospores**, small, swimming spores, each with a single, posterior **flagellum**. The word *zoospore* suggests that they look like "animal spores" because they are motile. These fungi survive as **resting sporangia** from which zoospores are later produced. Chytridiomycetes differ from other true fungi in having zoospores. They are often considered the most primitive group of the true fungi.

There are only a few plant-pathogenic chytridiomycetes, and all are biotrophs that are restricted to the cells of their hosts. Unlike other fungi, they produce motile zoospores and survive as resting sporangia.

ZYGOMYCOTA

ZYGOMYCETES

- **nonseptate** hyphae
- sexual spores: large, dark **zygospores**
- asexual spores: **sporangiospores**, produced in a **sporangium**

Most of the plant pathogens in this group are weak pathogens, usually of stored fruits and vegetables (as postharvest agents of decay) and propagative parts. The majority of zygomycetes are saprophytes. Some species of the zygomycete genera *Rhizopus* and *Mucor* are used in the commercial production of lactic acid and certain enzymes, as well as in the fermentation of soybeans to produce the Asian foods tempeh and tofu. Some fungi used for biological control of insects are found in the order Entomophthorales in Zygomycota. The arbuscular mycorrhizal fungi (AM fungi or endomycorrhizal fungi) are important mutualists associated with the roots of many plant species. They were once classified in the zygomycetes but are now in their own phylum, Glomeromycota.

The sexual spores of zygomycetes, **zygospores**, are generally large and dark and produced at the union of compatible hyphae (Figure 2.13A). Asexual spores are called **sporangiospores**. These spores are small and airborne when released from the fragile membrane-covered **sporangium** at the end of a hyphal stalk (Figure 2.13B). The hyphae are nonseptate (coenocytic). Like the conidial stages of ascomycetes, the sporangial states of zygomycetes are the most apparent form that occurs on affected plants, usually causing soft rots of fruits or flower blights.

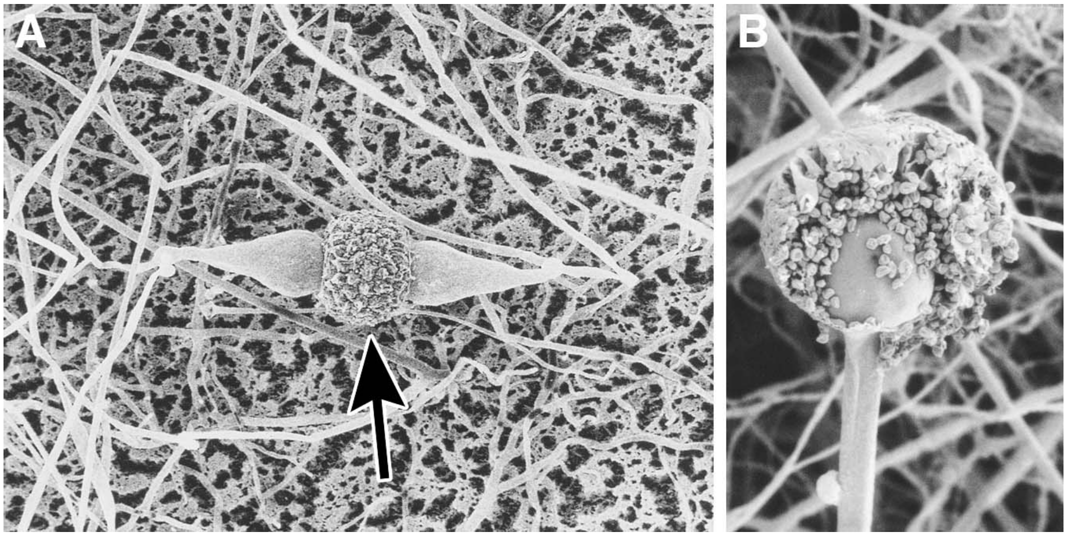


Figure 2.13. Zygomycete spores. A, Zygospore (sexual). B, Sporangium and sporangiospores (asexual).

Zygomycetes cause some postharvest diseases, but most growing plants are not susceptible to diseases caused by

zygomycetes. They are characterized by nonseptate hyphae, sexual zygospores, and asexual sporangiospores.

Plant Pathogens Formerly Classified as Fungi

The following fungus-like groups of organisms have evolutionary lineages different from those of true fungi, but they can be easily confused with them. Oomycota produce both hyphae and spores. Myxomycota and Plasmodiophoromycota produce spores. Disease symptoms caused by many members of these groups may be similar or identical to those caused by true fungi.

OOMYCOTA

OOMYCETES

- **nonseptate** hyphae
- cell wall containing cellulose (**no chitin**)
- **diploid nuclei**
- sexual spores: **oospores**, produced after contact between an oogonium and an antheridium
- asexual spores: **zoospores**, with two **flagella**, produced in a **sporangium**

Sometimes known as the "water molds" or oomycetes, this group is more closely related to the brown algae than to the true fungi. Although oomycetes produce filamentous hyphae that look superficially like those of the true fungi, their cell walls are composed of cellulose rather than the chitin found in true fungi. The hyphae are nonseptate (coenocytic). The **diploid** (2N) rather than the haploid phase of the life cycle is dominant. Oomycetes are common in soils and surface waters and include many important soilborne and airborne plant pathogens such as species of *Pythium, Phytophthora* (derived from the Greek words meaning "plant destroyer"), and the organisms that cause white rust and downy mildew diseases.

The oomycetes are named for the microscopic round **oospores** (Figure 2.14) that are produced in sexual reproduction and serve as thick-walled survival spores. Most species have adapted to living in moist environments by producing asexual **zoospores** with flagella that enable them to swim. Each oomycete zoospore has two flagella, one whiplash (smooth) flagellum pointed backward and one tinsel (hairy) flagellum pointed forward. These flagella are different from those produced by chytridiomycetes and the two fungus-like groups described below. In most oomycetes, zoospores are produced in a **sporangium**. A sporangium is a fragile, saclike structure that may serve as inoculum itself (direct germination), or its contents may convert to asexual zoospores (indirect germination), each of which could potentially cause an infection (Figure 2.15).

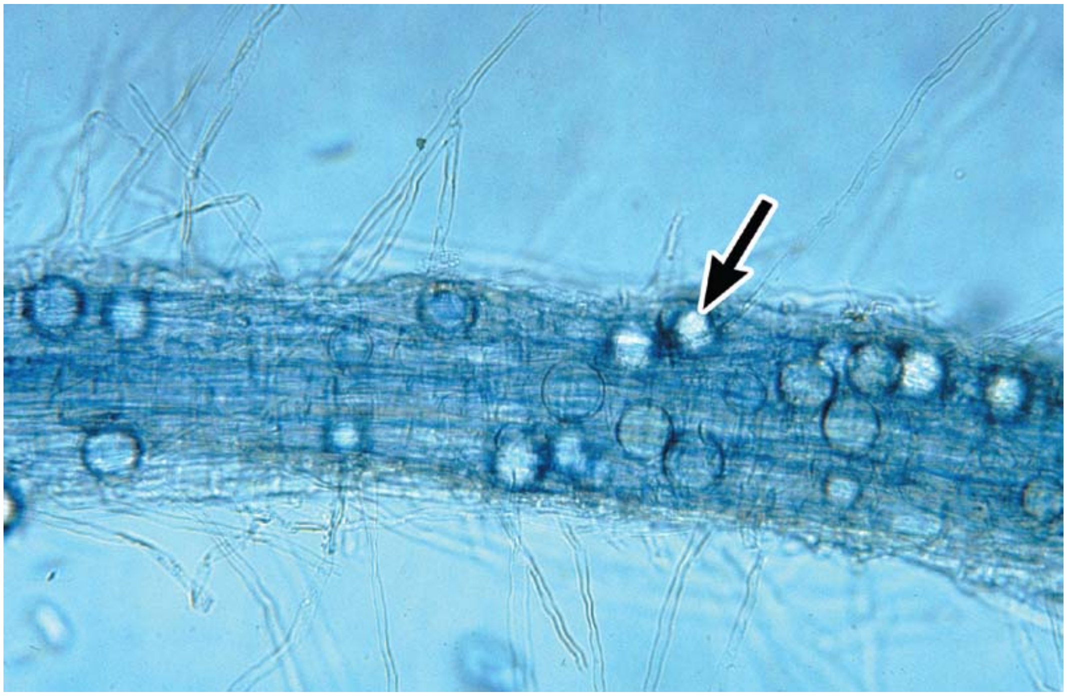


Figure 2.14. Thick-walled, sexual oospores (arrow) of *Pythium* sp. in a turfgrass root.

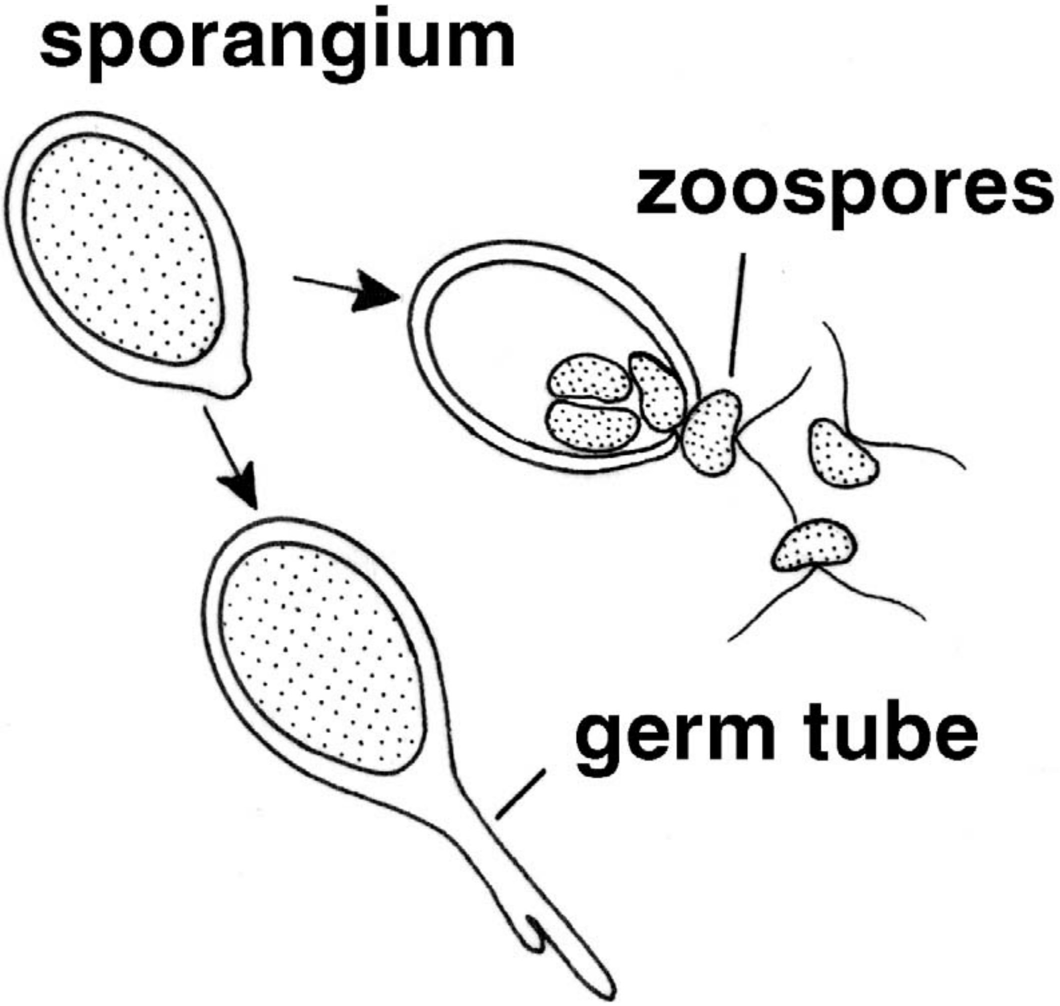


Figure 2.15. Sporangium of an oomycete producing asexual zoospores (indirect germination). Sometimes a sporangium functions as a single spore and produces a germ tube (direct germination).

Pythium spp. are found in all soils and are the most common cause of damping-off of seedlings under wet conditions. Most diseases caused by *Pythium* spp. and *Phytophthora* spp. are soilborne or occur in the "splash zone" between soil and lower plant parts. Many species have wide host ranges and are responsible for a variety of stem, root, and crown rots. An important exception is *Phytophthora infestans*, the species responsible for late blight of potato and an important factor in the Irish potato famine (see Chapter 1). Its sporangia are produced in a white fringe at the edge of active leaf lesions. The sporangia may be dispersed by water or air to new leaves or washed into the soil where they can infect tubers.

White rust (Figure 2.16) and **downy mildew** (Figure 2.17) are diseases caused by more specialized oomycetes that are biotrophs (obligate parasites), host specific, and adapted for air dispersal to aboveground plant parts. Even though they appear quite different from many oomycetes, they still produce oospores in sexual reproduction, and zoospores are produced by many species when the airborne sporangia land and germinate on moist leaves. White rusts are named for the pustules of white, powdery sporangia that look somewhat similar to the orange pustules produced by rust fungi, which are basidiomycetes. Downy mildew pathogens produce sporangia on specialized branched (treelike) hyphal structures (**sporangiophores**) that aid in the air dispersal of the sporangia. Sporangia of a number of oomycetes have the ability to produce zoospores or germinate directly as single spores, depending on environmental conditions. In their adaptation to a dry foliar environment, some downy mildew pathogens never produce zoospores at all. Their sporangia function as individual asexual spores and, because they are produced asexually, are sometimes referred to as conidia.

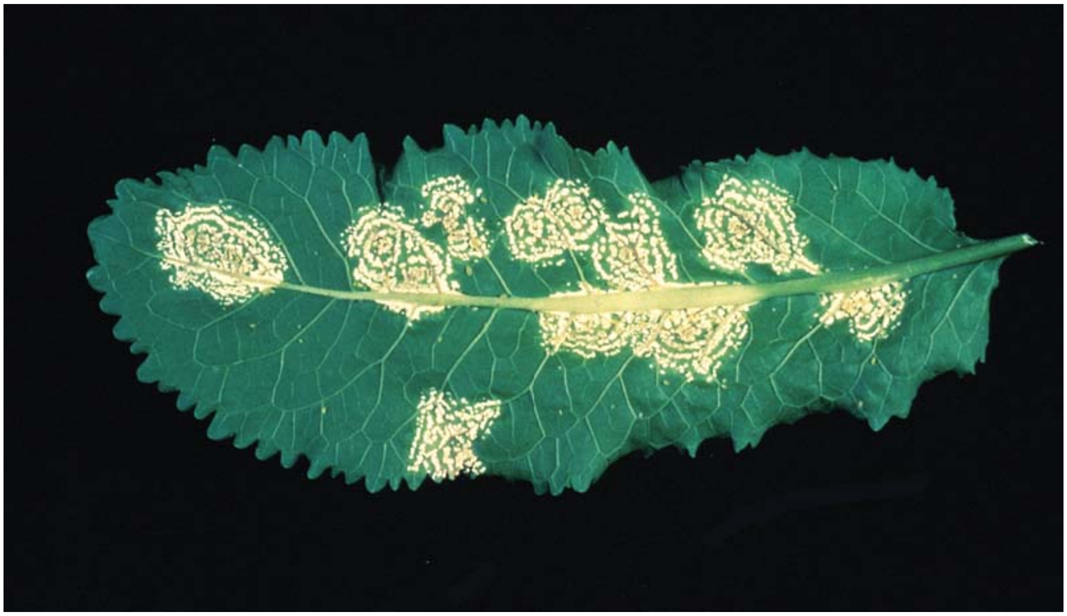


Figure 2.16. White rust (*Albugo candida*) on a brassica (crucifer) leaf.

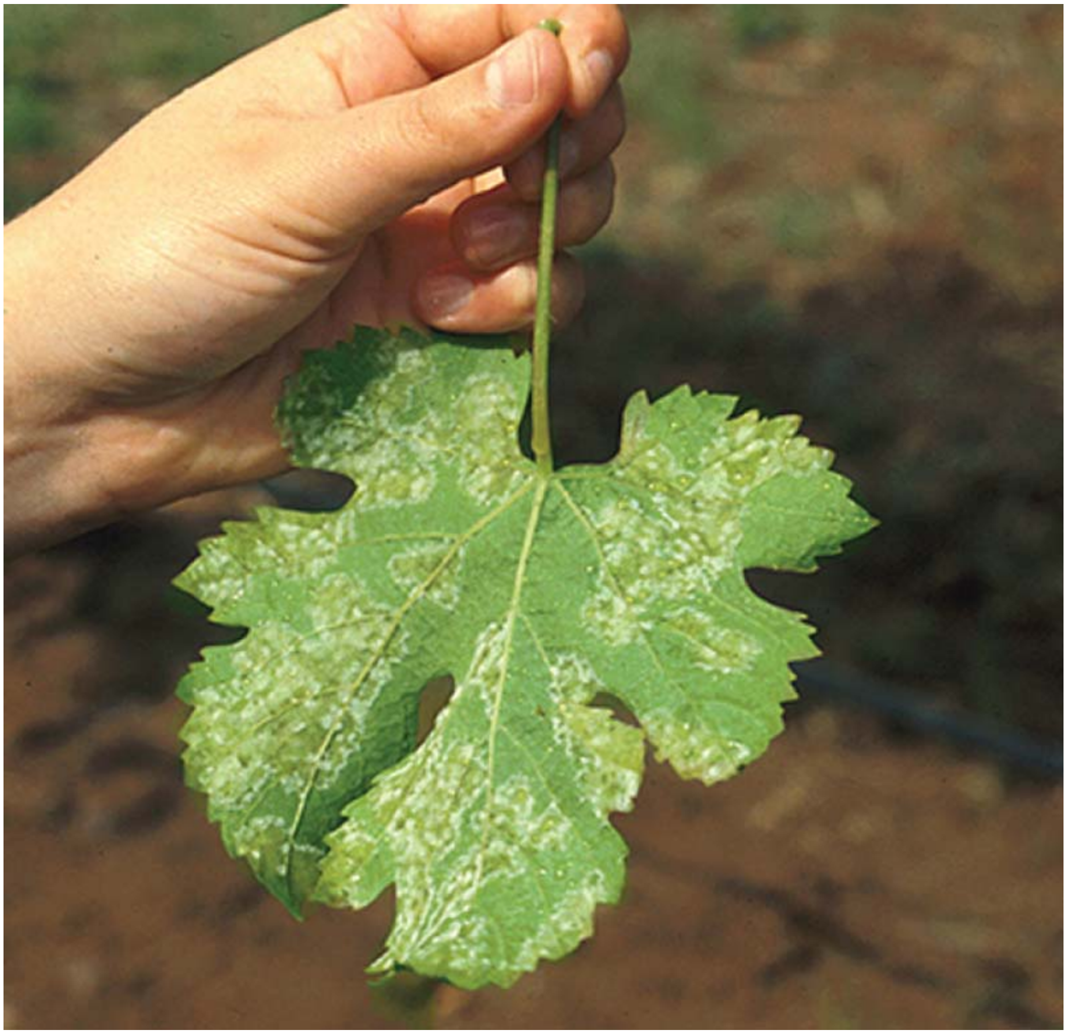


Figure 2.17. Downy mildew on the lower surface of a grape leaf. Note the sporangia and sporangiophores.

► DID YOU KNOW?

The Irish potato famine is well known for its impact on the poor in Ireland, who relied almost completely on potatoes for their food. Cereal crops were grown in Ireland and exported throughout the famine, because the money was needed to pay the rent on land owned by (mostly) English landlords. Although they were not as dependent on potatoes as the Irish, poor people throughout Europe suffered from the late blight epidemics during the “hungry 40s” of the 19th century. These epidemics may have contributed to the political climate that led to the publication of *The Communist Manifesto* in 1848.

Oomycetes form nonseptate hyphae, thick-walled oospores (sexual), sporangia, and swimming zoospores (asexual). Diseases caused by *Pythium* and *Phytophthora* species usually affect roots or the “splash zone” of plants. Some *Phytopththora* species are dispersed by air. White rust (caused by *Albugo* spp.) and downy mildews are diseases of aboveground plant parts. The pathogens that cause these diseases are biotrophs (obligate parasites), and their sporangia are adapted for air dispersal.

MYXOMYCOTA

Members of Myxomycota are commonly known as **slime molds** or myxomycetes. They seem more similar to fungi than to other kinds of organisms, as is reflected in the ending of the name, but biologically they are quite different. Their final classification remains to be decided, but they belong to an evolutionary lineage more closely related to that of protozoan animals than to either plants or true fungi.

Rather than hyphae, the vegetative stage of myxomycetes is a **plasmodium**, a mass of cytoplasm containing many nuclei but without a cell wall. Like an amoeba, the plasmodium feeds by phagocytosis, i.e., ingestion followed by digestion, rather than through the production of extracellular enzymes, as in true fungi. Reproduction is by **spores. Zoospores** with one to four flagella are produced. Slime molds are not plant parasites but can appear overnight during wet weather and cover grasses and leaves near the soil. The plasmodium actually moves from the soil onto these plant parts in an amoebalike ooze and is generally converted into a mass of spores in a day or so.

► **DID YOU KNOW?**

The sudden, and sometimes massive, appearance of slime molds was the basis of the 1958 science fiction movie *The Blob*.

See Tom Volk’s Fungi (website), Fungus of the Month, June 1999.

Myxomycetes form a plasmodium that eventually produces a mass of spores. They are not plant parasites. Because slime molds can appear suddenly near or on plants, they can cause concern for people who do not know that they are usually harmless.

PLASMODIOPHOROMYCOTA

Sometimes called the endoparasitic slime molds or plasmodiophoromycetes, these organisms form a plasmodium (a mass of cytoplasm containing many nuclei but no cell wall), as their name suggests, but the plasmodia are restricted to the cells of the host plants. It is not clear to which kingdom these organisms belong, but like the myxomycetes, they are more closely related to protozoan animals than to true fungi. During their life cycle, zoospores with two flagella are produced. At maturity, the plasmodium becomes a mass of resting spores that are released from the plant tissue as it decays. All species are biotrophs (obligate parasites) of plants, algae, and fungi. The most significant plant diseases in this group are clubroot of brassicas (crucifers), such as cabbage and broccoli (caused by *Plasmodiophora*), and powdery scab of potato (caused by *Spongospora*). Infection by *Plasmodiophora brassicae* causes root cells to expand and multiply to form galls, leading to the name *clubroot* (Figure 2.18).

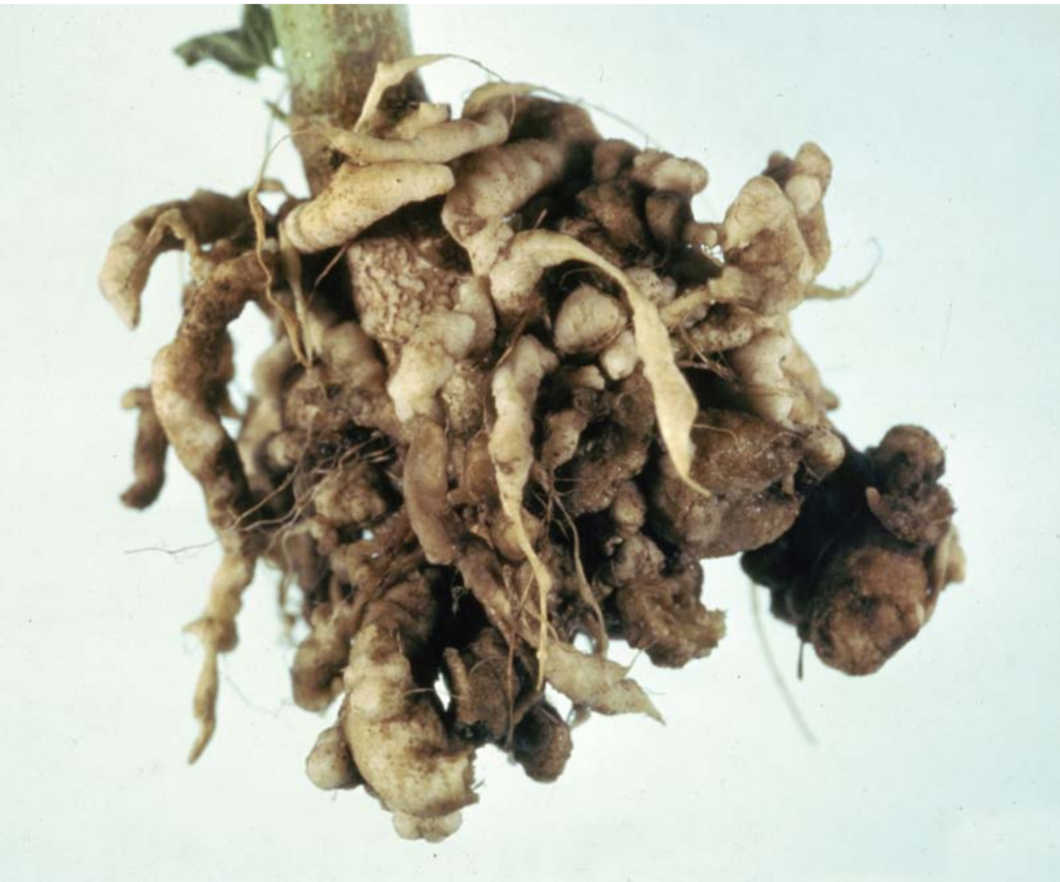


Figure 2.18. Clubroot of broccoli, caused by *Plasmodiophora brassicae.*

DISEASE CLASSIC

Clubroot of Brassicas (Crucifers)

Hosts most common brassica crops, including cabbage, broccoli, and cauliflower

Pathogen *Plasmodiophora brassicae*

Symptoms and Signs Above ground, plants are stunted and wilted, because of the clubs (galls) on the roots. Resting spores can be observed microscopically inside mature galls.

Disease Cycle The resting spores are released from decaying or damaged root galls. They germinate to produce zoospores, which infect root hairs of new host plants. A plasmodium forms in the infected root hair, becoming a mass of spores that releases a second set of zoospores. These zoospores fuse to form a cell capable of infecting root cells and causing gall formation. Each infected root cell is filled with a plasmodium that develops into a mass of resting spores at maturity.

Management In small plantings, destruction of infected roots before they decay and release the resting spores is helpful. In commercial plantings, soil pH of 7.2 or higher inhibits spore germination. Lime may be added to raise soil pH. Heavily infested fields must be planted to nonsusceptible crops because the pathogen can survive for many years and can even be distributed in manure if animals are fed infected plants. Genetic resistance is available for some races of the pathogen.

Significance *P. brassicae* is a destructive soilborne pathogen, found worldwide in areas where brassica (crucifer) crops are grown.

Plasmodiophoromycetes, like myxomycetes, form a plasmodium that eventually becomes a mass of spores. Unlike myxomycetes, the tiny plasmodia are restricted to the cells of their host plants. There are only a few economically important plant pathogens in this group, and all are biotrophs (obligate parasites).

How do fungi survive and spread?

Fungi are well adapted to survival when conditions are not conducive for growth. This is a particular advantage to parasites whose host plants may not always be available. Sometimes fungi and fungus-like organisms have thick-walled survival spores (e.g., oospores, resting spores, and zygospores). Some fungi produce fruiting bodies with melanized and thickened walls that help protect spores.

Not all survival structures involve spores. Sometimes the mycelium grows into a mass with a melanized rind of protective cells that helps prevent damage to the central mycelium. This structure is called a **sclerotium** (pl. sclerotia) (Figure 2.19). Sclerotia are produced by certain species of ascomycetes and basidiomycetes. They are often easily visible and vary in color from tan to brown to black. Individual fungal cells of a mycelium or even single cells of multicellular spores may develop thick walls and become **chlamydospores** (Figure 2.20), which are another type of asexual survival spore.

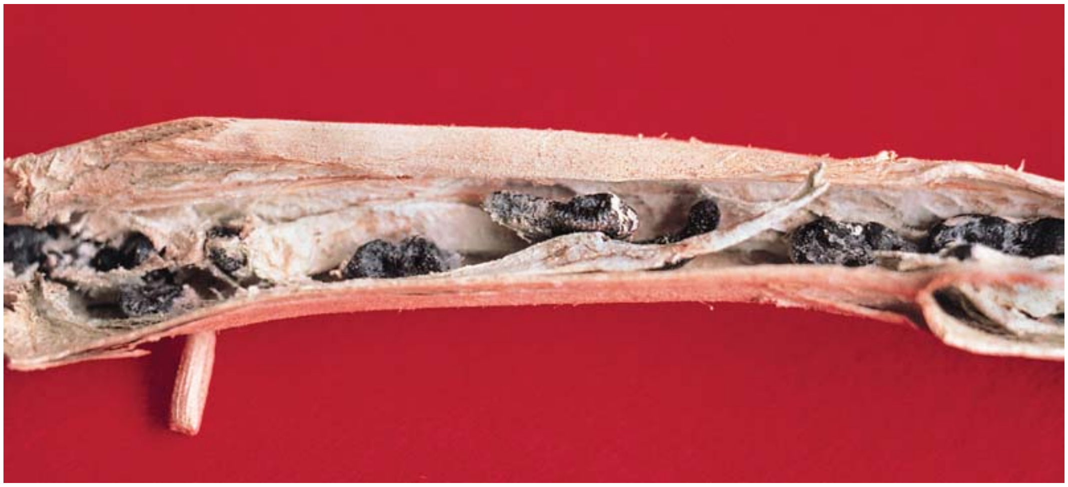


Figure 2.19. Large, black sclerotia of the white mold fungus (*Sclerotinia sclerotiorum*) in a tomato stem.

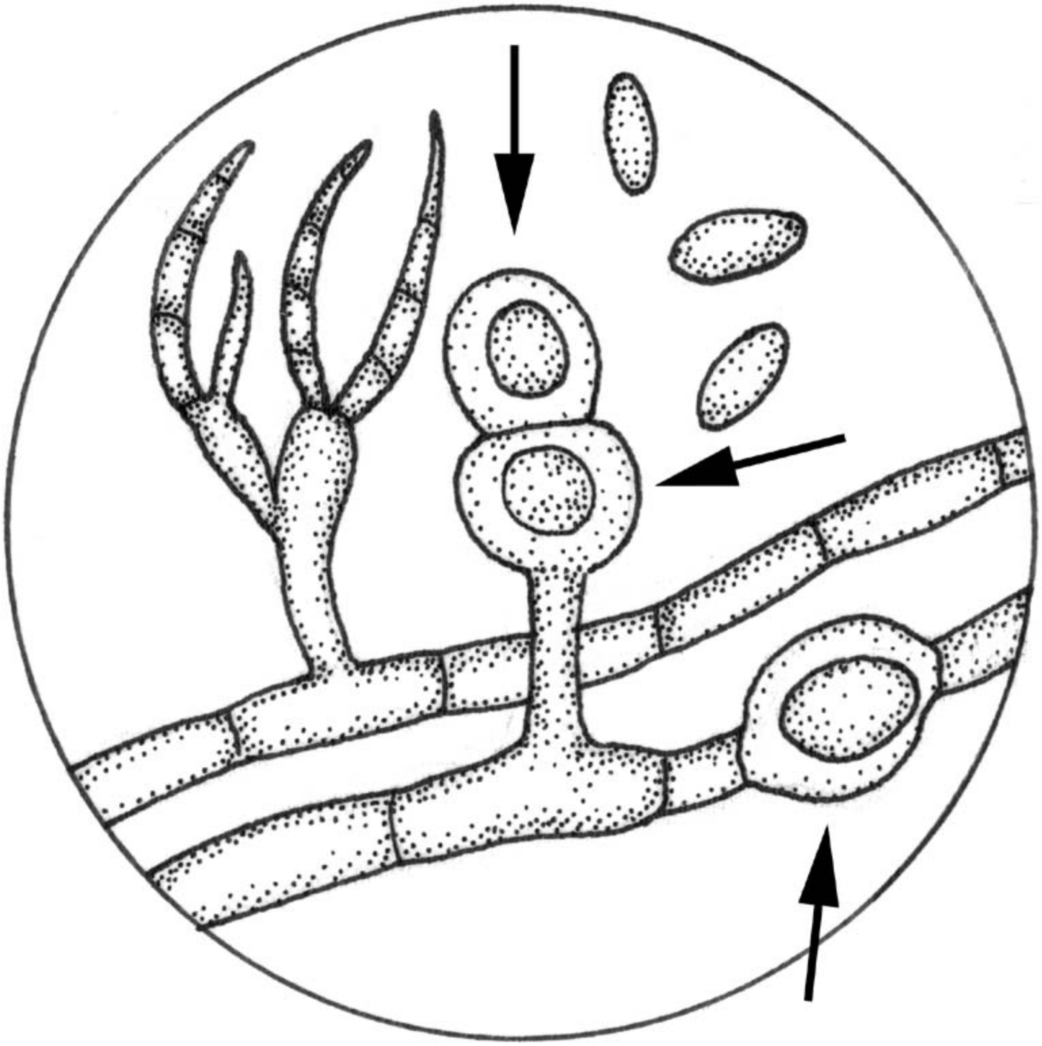


Figure 2.20. Thick-walled chlamydospores of *Fusarium* sp. (arrows).

Fungi survive by producing spores, fruiting bodies, chlamydospores, sclerotia, and other structures. Some survive as saprophytes in plant debris and soil. Most fungal spores are air-dispersed and may be forcibly discharged into the air, but others are sticky and dispersed by water, tools, and insects. Zoospores are adapted to dispersal by movement in water.

Parasitic fungi can coexist with perennial hosts, sometimes for years, as long as the host remains alive. Propagative parts, such as bulbs, corms, and tubers harvested from infected plants, are likely to carry spores, fruiting bodies, sclerotia, and/or mycelium of a plant-pathogenic fungus. Seeds may carry spores or hyphae on the seed coat (**infested** seed), or the embryo and cotyledons may be infected with mycelium (**infected** seed). Biotrophs can persist in infested plant debris only as survival structures. Facultative fungi are capable of both saprophytic and parasitic growth. They can continue to grow as saprophytes for some time in plant debris or, as the debris decays, in the soil, from which they may infect a new host. Even though they can exist as saprophytes, they also may produce structures to survive winter and other environmental extremes.

Spores are dispersed in many ways as well. Many spores are tiny and produced in huge numbers in a dustlike mass that is easily dispersed by the slightest air movement. Some fungi have adaptations that allow them to forcibly discharge their spores into the moving air surrounding the fruiting body on which they are produced. Ascospores and basidiospores are often discharged in this manner. Air dispersal is important for aboveground plant pathogens. It aids in the spread of the pathogen throughout a field or greenhouse and is important for long-distance dispersal.

Many spores are produced in sticky masses that are dispersed by splashing water and anything with which they might come into contact, such as machinery, tools, or passing insects. Even dry spore masses can be dispersed by splashing rain (somewhat like water droplets hitting and spreading talcum powder or chalk dust). This is due to the large surface area of spores relative to their tiny diameters. Zoospores are specifically adapted for swimming in water in the soil and on plant surfaces. A few fungi have specific relationships with insect vectors. One of the most famous is *Ophiostoma ulmi*, a fungus that causes Dutch elm disease. This ascomycete produces fruiting bodies topped with sticky spores in the larval galleries of elm bark beetles. The emerging adult beetles carry the spores to the next elm tree victim (see Disease Classic: Dutch Elm Disease). The survival and dispersal of fungi are discussed in more detail in Chapter 9.

How are fungal diseases diagnosed?

Some diseases such as powdery mildews, rusts, and smuts can be diagnosed by observing their characteristic spores on the host plant. For most common diseases, a combination of plant symptoms and pathogen signs is often sufficient to come to a diagnostic conclusion.

There are some significant limitations to this simple method. It may not be possible to clearly determine which of the fungi (if any) observed on the diseased tissue is the primary pathogen. Secondary invaders and saprophytic contaminants often grow under the same conditions as pathogens in a moist chamber. These same organisms may invade plants stressed by abiotic factors. One approach to solving this problem is to **disinfest** (surface sterilize) small pieces of infected tissue, typically by immersing them in dilute solutions (10–50%) of household bleach (sodium hypochlorite) for short periods of time (15–60 seconds depending on tissue type). This helps kill surface contaminants (secondary invaders and saprophytes). If the piece of tissue is left in the bleach solution too long, both the contaminants and the pathogen will be killed and the tissue will be **disinfected**. Note that table tops and tools may be disinfested, but not disinfected, because they are not infected in the first place.

The disinfested pieces of plant tissue are blotted dry and placed on a suitable fungal nutrient medium, such as potato-dextrose agar (PDA), in a petri dish (Figure 2.21). It is not possible to know exactly how long it will take to disinfest the plant tissue, but not disinfect it. The amount of time required in the bleach solution depends on the size and type of plant tissue and the extent of its deterioration (contamination). In most cases, small samples of tissue are placed in solution for a range of times (e.g., 0, 15, 30, 45, or 60 seconds) to increase the probability of successfully obtaining a clean isolate of the pathogen. Most fungal pathogens will grow out of the plant tissue and into the agar (Figure 2.22).

► **DID YOU KNOW?**

Agar is a polysaccharide derived from red algae that serves as a moist gel on which fungi can grow. Most fungi do not metabolize the agar, so we add other components, such as potatoes and dextrose for the common medium potato-dextrose agar (PDA), to provide the nutrients needed by many fungi. To enhance or restrict growth of certain organisms, antibiotics, pieces of host tissue, or other components may be added or withheld. Media must be sterilized in a pressure cooker or autoclave. Agar melts at 100°C (212°F) and solidifies at about 45°C (113°F).

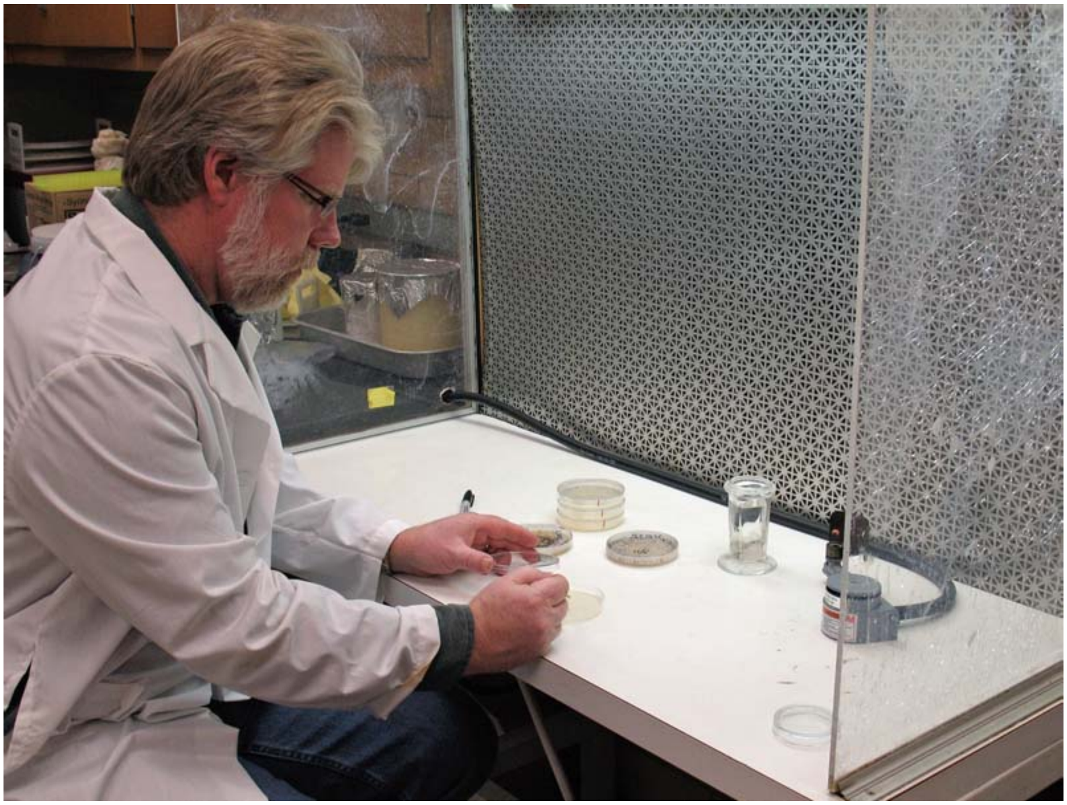


Figure 2.21. Plant pathologist working in a laminar flow hood and using sterile tools to isolate and transfer fungi in petri dishes.

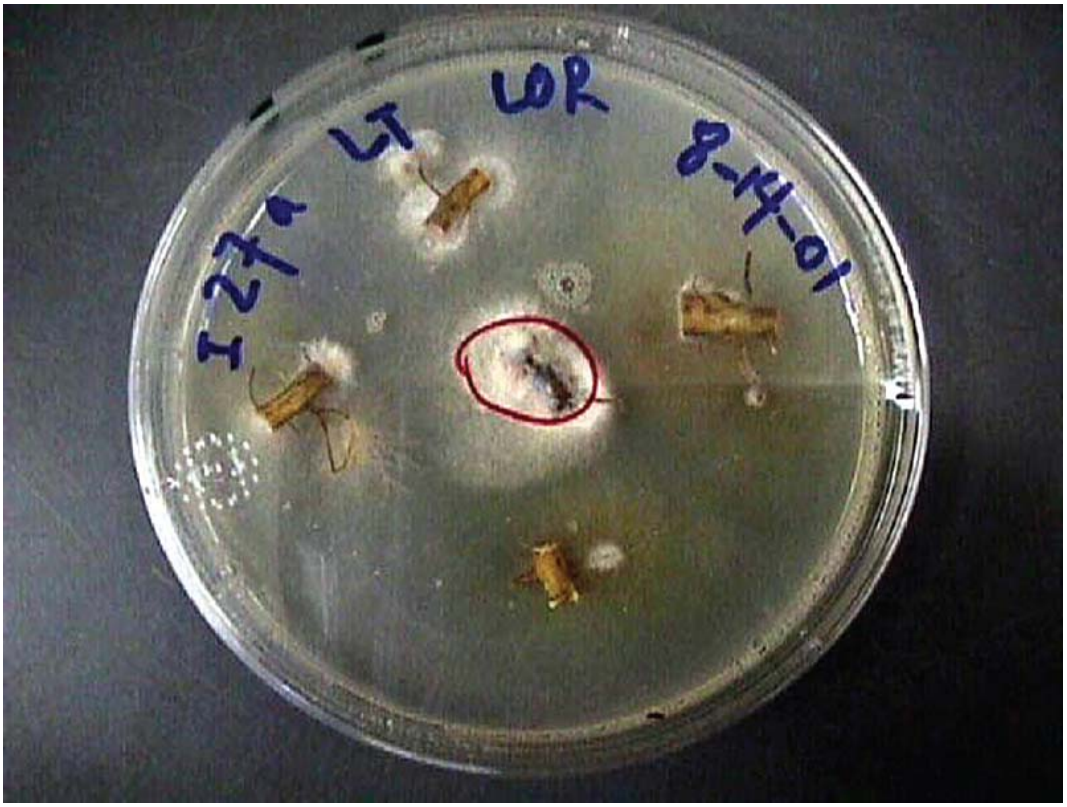


Figure 2.22. Isolation of a fungal pathogen from plant tissue. Note variable growth.

This method cannot be used for biotrophs (obligate parasites), which grow only on living hosts or highly specialized media, but it works well for most facultative organisms. It is common to recover several different species of fungi. Spores and fruiting bodies necessary for identification of the fungi may develop quickly, or, unfortunately, they may take weeks or months to develop. Sometimes two different mating types of the fungus are needed for sexual stages to form (**heterothallic fungi**), and both may not be present. If the plant tissue is badly deteriorated, the primary pathogen may no longer be present, and only secondary invaders or saprophytes will be isolated. These limitations are one reason why Koch’s postulates must be fulfilled for new diseases. They also help explain why diagnostic labs may not be able to complete a diagnosis quickly.

Modern rapid methods for identification of true fungi and fungus-like organisms are based on immunoassays (antibody tests) or use of the polymerase chain reaction (PCR) to identify specific DNA sequences. (See Appendix 3 for detailed information about these diagnostic methods.) These methods are particularly helpful when one is trying to identify fungi and fungus-like organisms that do not readily grow in culture (e.g., biotrophs) or do not produce spores. The tests also are useful for economically important pathogens for which rapid identification is essential. Some immunoassays have been developed as field kits that can be used without special equipment, but they tend to be expensive and have a limited shelf life. PCR diagnostic tests are generally performed in diagnostic labs for a fee.

Diagnosis of a fungal plant disease requires identification of the fungus. Look for spores and fruiting bodies. Moist chambers enhance the growth of mycelium and spore production. If diagnosis is uncertain or Koch's postulates must be fulfilled for a new disease, the pathogen must be isolated into pure culture. Disinfest some infected tissue and place it on a nutrient medium such as potato-dextrose agar (PDA).

► DID YOU KNOW?

Coffeehouses were common in Egypt, Arabia, and Turkey by the early 1500s. The Dutch became the primary suppliers for Europe by planting coffee in their colonies in Ceylon, Sumatra, and Java. *Java* remains a slang term for coffee in many places. After taking control of Ceylon, the British expanded the coffee plantations and produced nearly 100 million pounds of coffee a year, mostly for England. By 1879, coffee rust was destroying the coffee plantations. Coffee rust develops from urediniospores only. Tea, for which Ceylon (Sri Lanka) is now famous, eventually replaced the failing coffee crop, and drinking tea became a social institution in England. Coffee rust was excluded from the Western Hemisphere for nearly 100 years by quarantines but is now a worldwide disease.

How are fungal diseases managed?

Knowledge of the disease cycle associated with a particular fungal pathogen is essential for deciding on a management plan. The following methods are used to manage fungal diseases and are discussed in more detail in later chapters.

Management Strategies for Fungal Diseases

Avoidance

- Choose planting sites and planting times to avoid environmental conditions favoring disease.

Exclusion

- Impose quarantines (local and international).
- Plant pathogen-free seed or stock.

Eradication

- Rotate to nonhost crop.
- Remove weed hosts.
- Destroy infested plant debris.
- Apply fungicides.

Protection

- Apply fungicides.

- Minimize leaf wetness.
- Plant resistant hosts.

Avoidance

Some fungal diseases can be prevented by choice of planting site or timing of planting.

Exclusion

Both international and local quarantines are used to protect plants from fungi that are not yet present in certain areas. Production of pathogen-free planting material is important for excluding fungi that are easily spread during vegetative propagation.

Eradication

The pathogen population can often be significantly reduced by removing infested debris or by planting nonsusceptible plants in the infested soil for one or more years (crop rotation). Cankers and other localized problems can be removed from woody plants by pruning to prevent spread of spores to new infection sites and neighboring host plants (Figure 2.23).

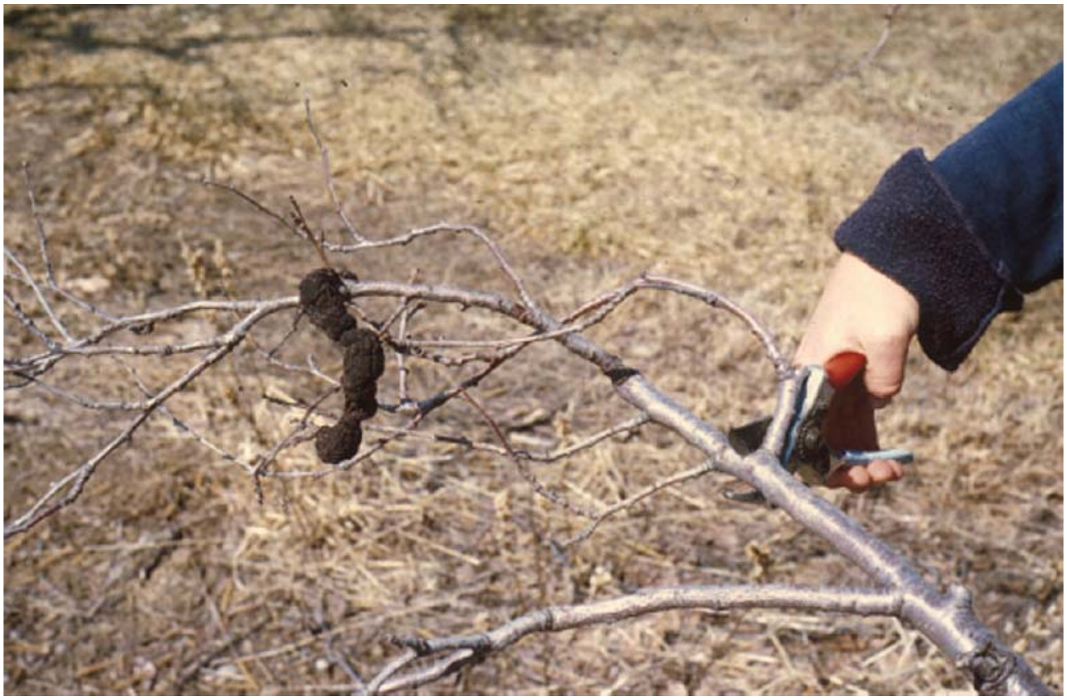


Figure 2.23. Eradication: pruning black knot cankers from a cherry tree.

Protection

There are many cultural practices that can help protect plants from fungal infections. If the fungus produces sticky spores, using tools and touching the plants, especially under wet conditions, can increase spore dispersal. If the spores are airborne, they may arrive from distant areas, which is often unavoidable. However, spores landing on aboveground surfaces usually require free water for germination and infection. Thus, various methods for minimizing leaf wetness have been developed, including timing of irrigation, placement of irrigation equipment, plant spacing, and row orientation to optimize air movement and speed drying of leaves.

To protect high value crops or to protect any crop from destructive diseases, fungicides may be applied to protect plants against infection (Figure 2.24). Most fungicides are strictly protective and cannot arrest an infection once it has been established. Some fungicides can penetrate plant tissues and stop infections in their early stages, but they are still primarily protective in their activity. Plant cultivars (varieties) with genetic resistance to specific fungal diseases have been developed for a wide variety of crops. The use of resistant cultivars is especially important when the cost of fungicide protection is prohibitive or when good chemical treatment options do not exist. Fungal adaptation is a continual challenge to disease management with both fungicides and genetic resistance. Cultural practices that promote plant vigor and reduce environmental stresses (predisposition) also reduce the plant's susceptibility to disease. They enhance the effects of genetic resistance and improve the plant's response to chemical treatments.

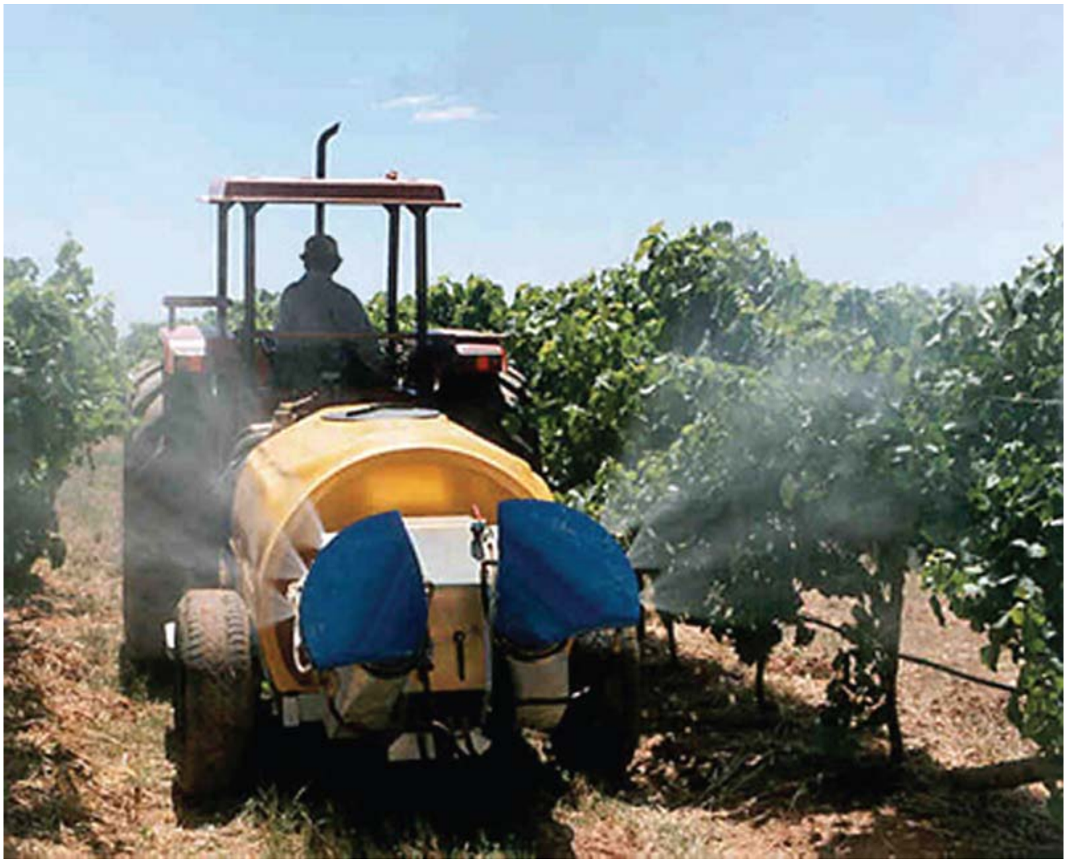


Figure 2.24. Protection: fungicide application in a vineyard.

The choice of appropriate management practices depends on the pathogen, the host, and the environment. These are all discussed in more detail in the chapters that follow.

DISEASE CLASSIC

Downy Mildew of Grape

Hosts all varieties of grapes (*Vitis* spp.); *Vitis vinifera* (wine grape) is more susceptible than *Vitis lambrusca*, the American grape

Pathogen *Plasmopara viticola* (an oomycete)

Symptoms and Signs Yellow, circular, oily-looking spots appear on the upper leaf surface, each eventually developing a brown margin; on the lower leaf surface and on fruit, the white sporulation (downy mildew) of the pathogen appears in moist weather.

Disease Cycle The pathogen survives as oospores in infested debris. Sporangia are produced by the germinating oospores, which, in turn, produce zoospores. The sporangia and zoospores (primary inoculum) are airborne or splashed to lower leaves. Additional sporangiophores and sporangia form following the primary infections, resulting in the production of secondary inoculum. Sexual reproduction occurs toward the end of the growing season to form oospores for survival to the next season.

Management Cultural practices such as spacing, row orientation, and pruning reduce moisture on leaves and fruit and thereby reduce infections. Genetic resistance is difficult to combine with some desirable grape characteristics but is a focus of management strategies. Many grape cultivars must be protected with fungicides in moist climates.

Significance Grape downy mildew is a potentially destructive disease in almost all grape-growing areas under disease-conducive conditions. It nearly destroyed the French wine industry beginning around 1878 before Millardet developed the first foliar fungicide, Bordeaux mixture (copper sulfate + lime).

Plant Disease Profiles: Downy mildew of grape

Recall the disease triangle; management of fungal diseases of plants can involve reducing the pathogen population, modifying the environment to be less favorable to the pathogen or more favorable to the host, and protecting the plant with fungicides or through genetic resistance. Integrated approaches are usually most successful.

Questions

1. If you observe septate mycelium, to what fungal group or groups might the organism belong? What is one fungal or fungus-like group to which it does not belong?
2. Many fungi have a scientific (Latin) name for their teleomorph and one for their anamorph. The teleomorph name is also the name of the whole organism (holomorph). If both stages are known and named, why are anamorph names still used?
3. What are the names of the asexual and sexual spores for the following fungal and fungus-like groups? Complete the chart.

	asexual spores	**sexual spores**
ascomycetes		
basidiomycetes		
zygomycetes		
oomycetes		

4. Oomycetes cause a large number of significant plant diseases, but they are no longer classified as fungi. What are two

biological characteristics of members of this group that separate them from the "true" fungi?

5. Koch's postulates are the basis for proving pathogenicity of an organism found in association with a diseased plant (see Chapter 1). Find two pathogens in this chapter and describe how you would have to modify the standard approach to fulfilling Koch's postulates. (Hint: Look for biotrophs or diseases that develop under unusual environmental conditions or during particular stages of plant development.)

► **DID YOU KNOW?**

All blue and green cheeses—including English Stilton, French Roquefort, and Italian Gorgonzola—are produced by inoculation with conidia of the ascomycete *Penicillium roquefortii*. The genus *Penicillium* is named for its brushlike conidia and conidiophores (see Figure 2.29). It includes *P. notatum*, an important source of the antibiotic penicillin. Other *Penicillium* species are important postharvest pathogens of stored grain and apples.

To read more about the history of penicillin and the fungi that produce it, see Tom Volk's Fungi (website), Fungus of the Month, November 2003.

Challenge Questions

1. You have identified the causal fungus of a plant disease by observing its spores and fruiting bodies.
    a. Using what you know about fungi, do you think that a fungicide application at this point would be effective in stopping the spread of the disease?

    b. Using what you know about the life cycles of fungi, do you think that a plant cultivar will remain resistant to a pathogen if it is planted in the same area over many years?
2. Pythium root rot and Phytophthora root rot can threaten nearly any type of plant. Choose a plant of interest (for example, turfgrass, rhododendron in a landscape, geraniums in a greenhouse, squash vegetable crop), and describe how you could avoid these diseases.

More detailed information about fungi, their reproduction, and fruiting bodies

Ascomycota

ASCOMYCETES

- **septate** hyphae
    - **haploid** nuclei
- sexual spores: **ascospores**, formed in an **ascus** (sac)
- asexual spores: **conidia**

Ascomycetes have septate hyphae that contain haploid nuclei. The number of nuclei between each septum is variable. During sexual reproduction in ascomycetes, the hyphae of two sexually compatible individuals fuse. Haploid nuclei (one from each parent) pair up, divide at the same time for a short period, and, in a specialized cell, finally fuse to form a diploid nucleus. There is a delay between plasmogamy (the placement of two compatible nuclei in the same cell) and karyogamy (nuclear fusion) (Figure 2.25). During that time, the pairs of nuclei divide several times, forming a limited dikaryotic mycelium in which each cell contains the two sexually compatible haploid nuclei. After karyogamy, the resulting diploid nucleus undergoes meiosis to form four haploid nuclei. Typically, these nuclei divide once more, by mitosis, to form eight nuclei. Formation of cell walls around these nuclei results in a set of eight ascospores in an ascus (pl. asci), or sac (Figure 2.25). In most species, the asci have a single cell wall (**unitunicate**), but some species have a double wall (**bitunicate**). Usually, numerous asci are formed during sexual reproduction. The asci of some pathogens (e.g., *Taphrina* spp.) are naked (not in a fruiting body) on the surface of an infected plant, but they are most often produced in or on a fruiting body.

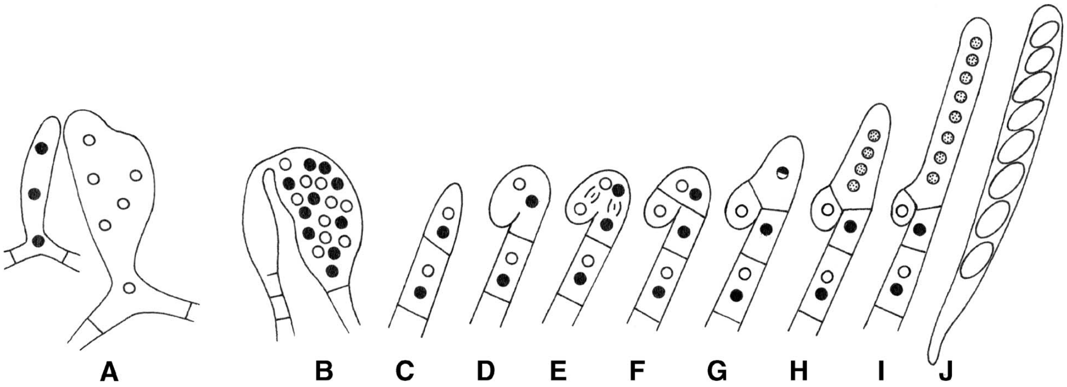


Figure 2.25. Generalized process of ascus formation. A, Male structure (often an antheridium) and female structure (ascogonium). B, Plasmogamy: the male structure contributes nuclei to the female structure. C, A limited dikaryotic mycelium develops. D–F, Through a complex mechanism, the ascus mother cell forms. G, Karyogamy: the two nuclei fuse to form a single diploid nucleus. H, Meiosis produces four haploid nuclei. I, In many ascomycetes, mitosis produces eight haploid nuclei and the ascus elongates. J, A spore wall forms around each nucleus to produce eight ascospores.

Sexual fruiting bodies. The general term for a sexual fruiting body that contains asci and ascospores is an **ascocarp** or an **ascoma** (pl. ascomata). Specific types include the following:

1. Fruiting bodies with **unitunicate** (single-walled) asci:

- **apothecium** a fleshy, cup-shaped fruiting body, usually produced from mycelium or on an overwintering structure (Figure 2.26A)

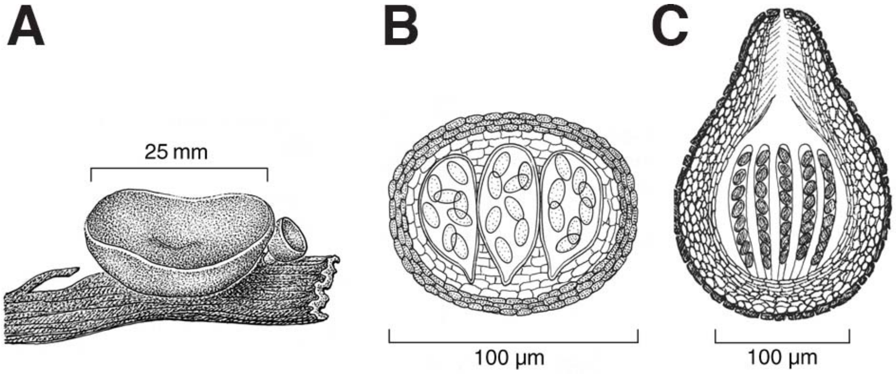


Figure 2.26. Ascomata with unitunicate asci. A, Apothecium. B, Chasmothecium (cleistothecium). C, Perithecium. Note the asci and ascospores.

- **chasmothecium** a closed fruiting body, typical of the powdery mildew fungi (Figures 2.26B and 2.27). ("Cleistothecium" is the term formerly used for one of these structures and is still found in many publications).

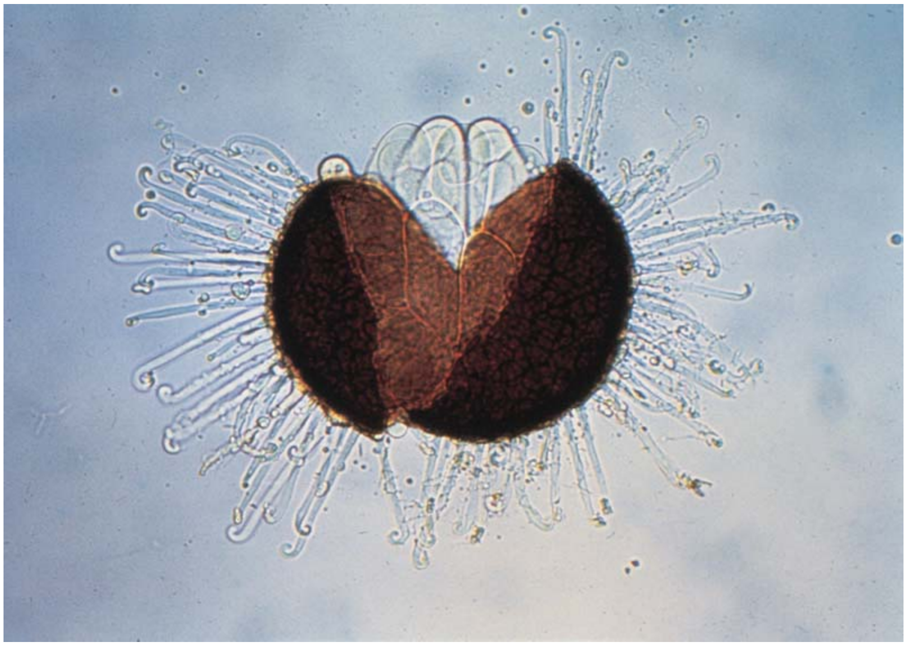


Figure 2.27. Chasmothecium (cleistothecium) of a powdery mildew fungus opening to reveal asci and ascospores.

   - **perithecium** a flask-shaped fruiting body, usually dark, with an opening (ostiole) for spore release (Figure 2.26C)
2. Fruiting bodies with **bitunicate** (double-walled) asci:
   - **ascostroma** a usually dark mass of mycelium containing cavities (locules) filled with bitunicate asci (Figure 2.28A)

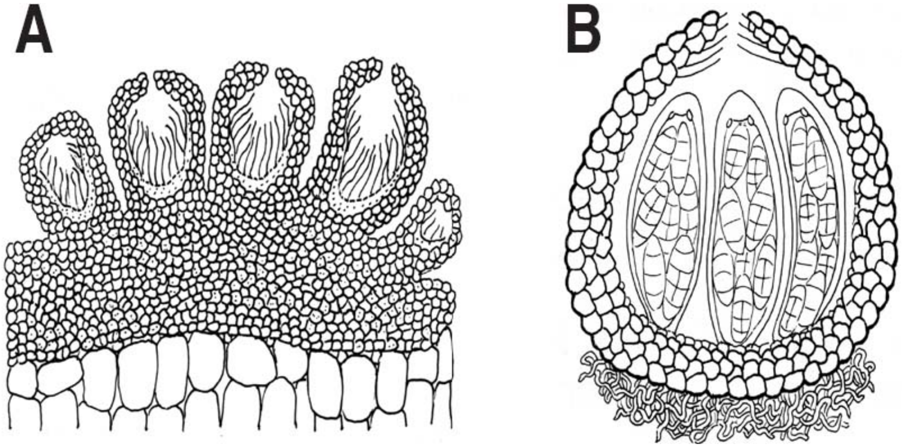


Figure 2.28. Ascomata with bitunicate asci. A, Ascostroma with multiple locules filled with asci. B, Pseudothecium. Note the double wall of the bitunicate asci.

   - **pseudothecium** a fruiting body very similar in appearance to a perithecium, but the asci are bitunicate (Figure 2.28B)

Asexual fruiting bodies. A generic term for an asexual fruiting body is a **conidioma** (pl. conidiomata). Asexual stages of fungi are often divided into two groups.

1. **Hyphomycetes.** Conidia are produced on specialized hyphal branches (conidiophores) not covered by a layer of fungal or host tissue. Conidiophores can be distributed evenly throughout the fungal colony (Figure 2.29), or they can be clustered together in one of these forms:
   - **sporodochium** a discrete cluster of conidiophores clumped together, often on a mass of sterile hyphae (Figure 2.30A)
   - **synnema** a “stalk” of conidiophores aggregated and fused together that raises the conidia above the surface of the substrate (Figure 2.30B)

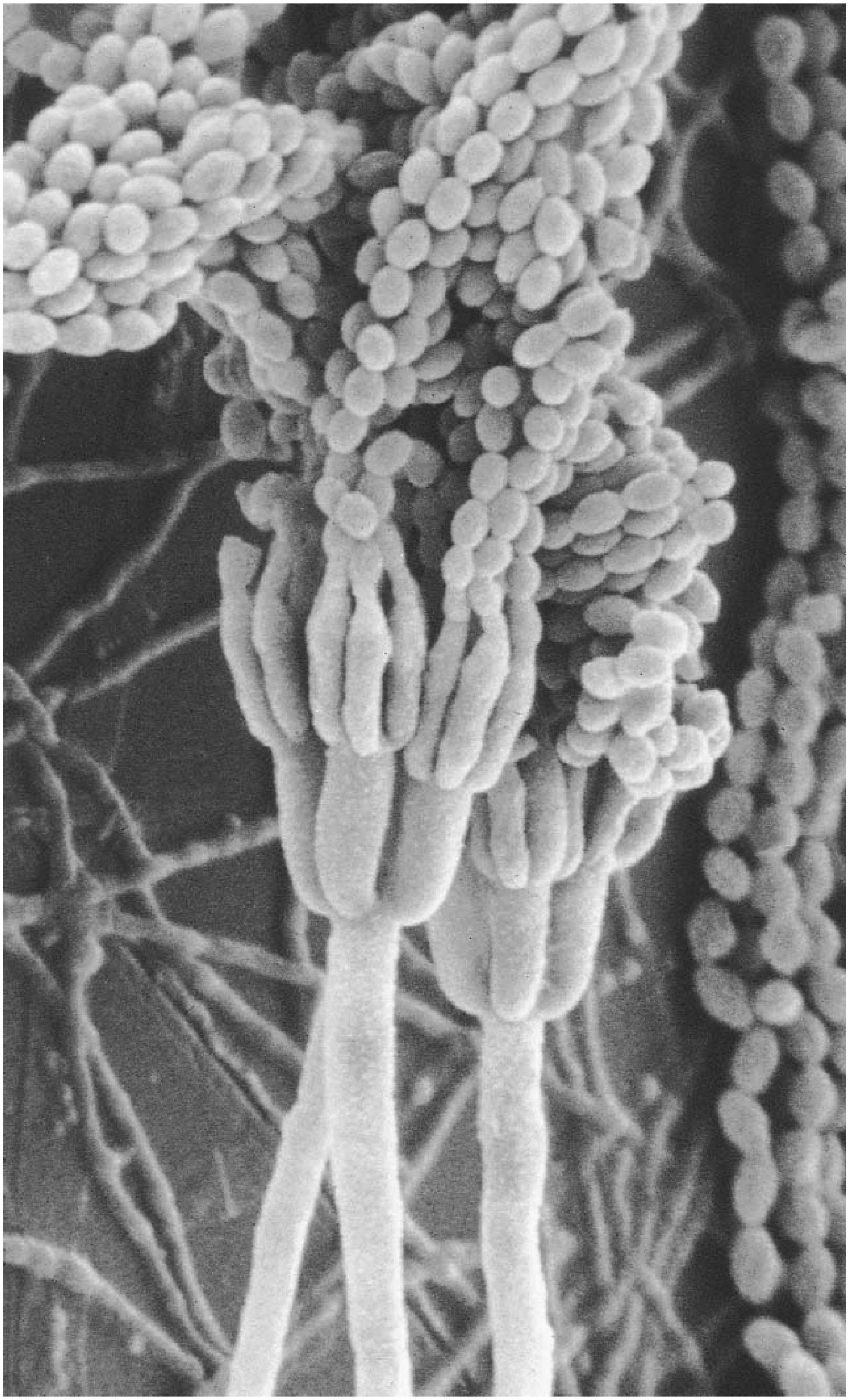


Figure 2.29. Conidia on a conidiophore (*Penicillium* sp.).

2. **Coelomycetes.** Conidia are formed from conidiophores within different types of fruiting bodies enclosed by a layer of fungal and/or host tissue:
    - **acervulus** conidiophores arranged in a thin layer below the host epidermis, becoming exposed by erupting through the plant epidermis, sometimes containing whiskerlike hyphae (**setae**) (Figure 2.30C); characteristic of anthracnose diseases
    - **conidial stroma** (pl. stromata) a structure similar to a pycnidium, in which conidiophores line the surfaces of multiple cavities (locules)
    - **pycnidium** a flask-shaped fruiting body with one or more ostioles (openings) in which conidiophores and conidia are produced (Figure 2.30D). This structure can look almost identical to a perithecium or a pseudothecium (both sexual fruiting bodies), but conidia are not produced in asci (sacs) as ascospores are.

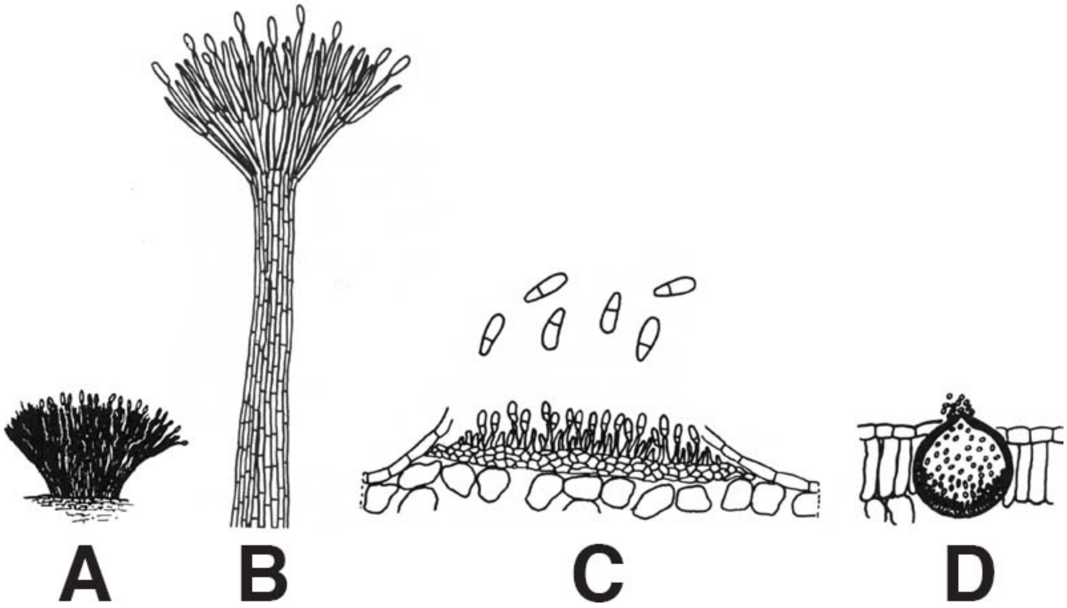


Figure 2.30. Conidiomata. A, Sporodochium. B, Synnema. C, Acervulus. D, Pycnidium. Note that the conidia are not in asci.

Note that several kinds of fruiting bodies look very similar in structure, e.g., chasmothecia (cleistothecia), perithecia, pseudothecia, and pycnidia. It is usually not possible to determine whether a spore is an ascospore or a conidium simply by observing it microscopically. The origin of the spores and other aspects of the fruiting bodies associated with them must be examined under the microscope.

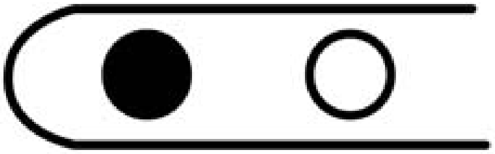
Fruiting bodies from infected plant tissue or culture plates can be observed by placing them on a microscope slide in a drop of water covered with a cover slip. Gentle pressure is applied to the fruiting bodies by pressing on the cover slip with a probe to see what kinds of spores can be squeezed out. Sometimes, ascospores in an ascus emerge. If the ascus is unitunicate, the fruiting body is a perithecium. If the ascus is bitunicate, the fruiting body is a pseudothecium. Sometimes free spores emerge. They may be conidia from a pycnidium. The asci of some ascomycetes dissolve after the ascospores mature, so some ascospores may fool you. With mature structures, caution is required to determine whether free spores are conidia or ascospores that have escaped from their ascus.

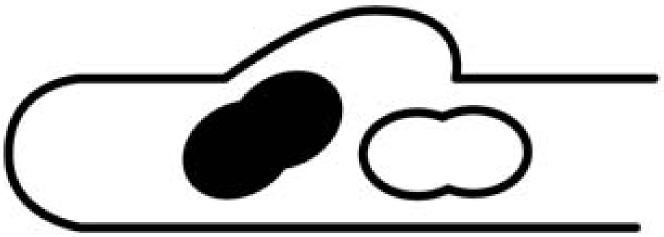
Basidiomycota

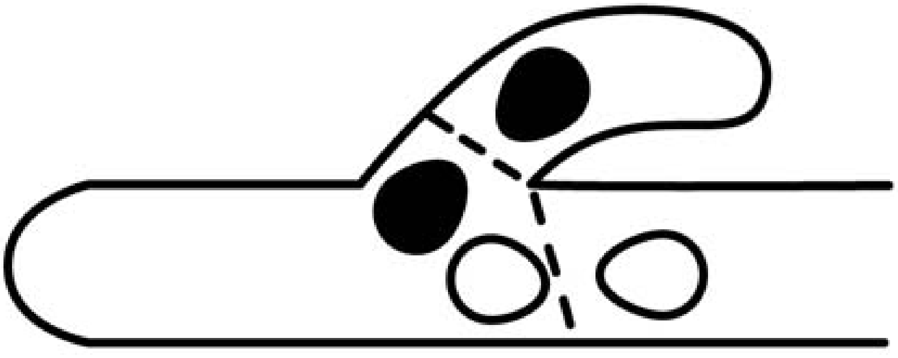
BASIDIOMYCETES

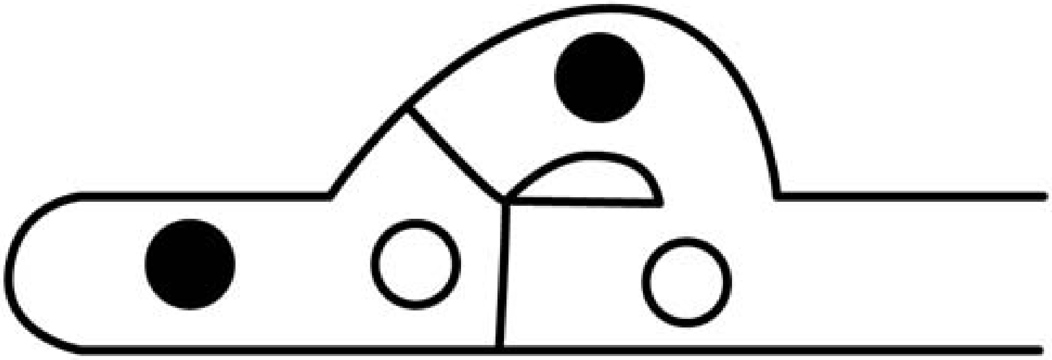
- **septate** hyphae (**some** species have **clamp connections** at some septa)
    - hyphae usually **dikaryotic**
    - **haploid** nuclei
- sexual spores: **basidiospores**, generally produced in a group of four external to the **basidium** (the cell that produces them)
- asexual spores: vary considerably within this group

Most basidiomycetes are **dikaryotic** for most of their lives. They have a pair of sexually compatible haploid nuclei in each cell that divide at the same time during mycelial growth. As in ascomycetes, this process begins when two sexually compatible haploid hyphae fuse to form the dikaryotic hyphae (plasmogamy). Although this stage lasts for only a limited time during the sexual reproduction of ascomycetes, it is the dominant vegetative state in most basidiomycetes. Each nucleus contributes to the genetic makeup of the organism, but they are not diploid because the nuclei have not fused. Clamp connections, which are a diagnostic structure of basidiomycetes, are produced only in dikaryotic hyphae. They are a specialized adaptation for maintaining the dikaryotic state as the mycelium grows (Figure 2.31). During mitosis, one of the duplicated nuclei travels through the clamp connection to the other side of the septum. Clamp connections are not required to maintain the dikaryotic state. Many dikaryotic basidiomycetes, including rust and smut fungi, do not have clamp connections.









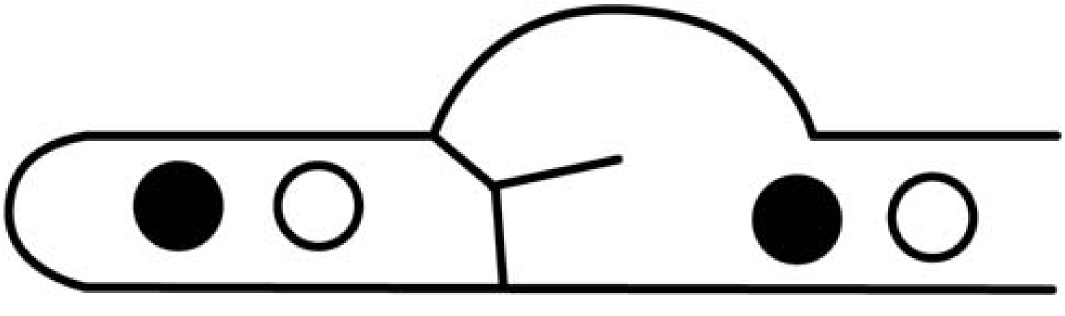


Figure 2.31. Clamp connection and dikaryotic hypha formed during mitosis.

In sexual reproduction, the two haploid nuclei fuse (karyogamy) in a specialized cell, the **basidium**, to form a diploid nucleus. As with ascomycetes, the diploid state is of brief duration. The diploid nucleus undergoes meiosis to produce four haploid nuclei, which migrate into the resulting four externally produced **basidiospores** (Figure 2.32A). In most cases, the basidiospores germinate to form a haploid mycelium, which, in various ways depending on the species, must fuse with a compatible haploid mycelium or spore to reestablish the dikaryotic state (plasmogamy). Rather than fusing, pairs of nuclei divide at the same time to form a mycelium that remains dikaryotic until the production of the next set of basidiospores.

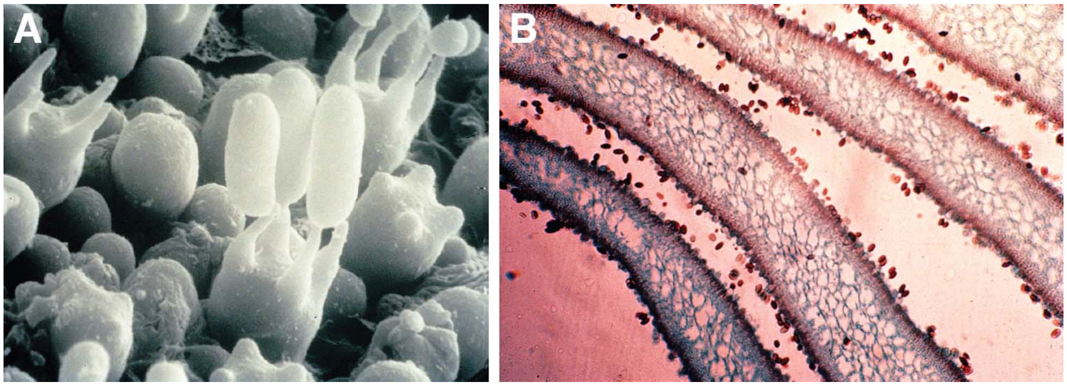


Figure 2.32. A, Four basidiospores on a basidium. B, Basidia and basidiospores along the gills on the lower surface of a mushroom.

As basidiomycetes, **rust fungi** produce haploid **basidiospores**. Their life cycle then becomes quite complex with up to five separate spore stages, each with a specific function (Figure 2.33). The basidiospores germinate, infect, and form **pycnia**, which are specialized haploid structures composed of **pycniospores** (also called spore stage **0**) and **receptive hyphae** that facilitate the pairing of compatible nuclei. This step is critical because it leads to the formation of the **dikaryotic** mycelium that produces the remaining spore stages.

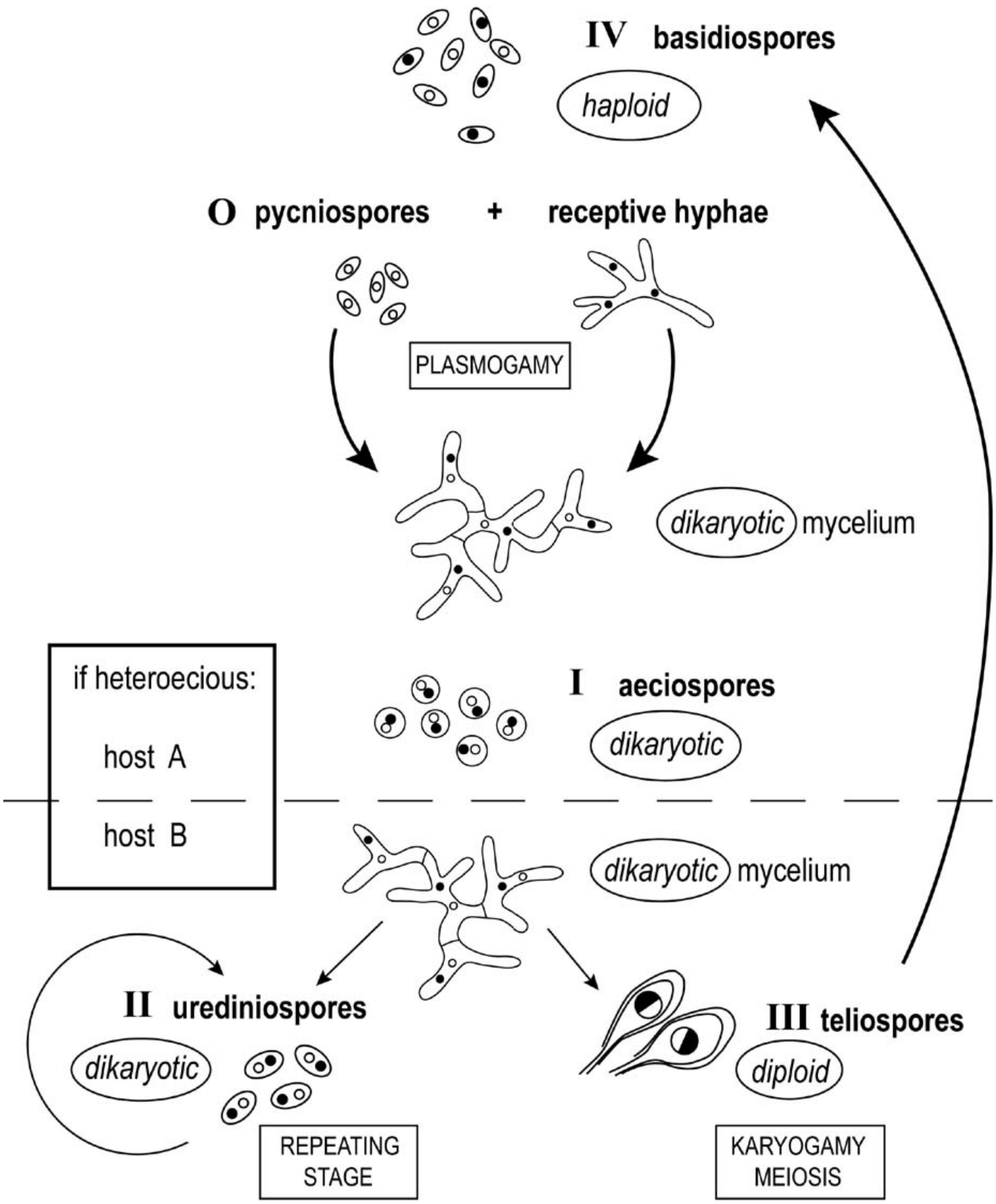

IV basidiospores
haploid
O pycniospores + receptive hyphae
PLASMOGAMY
dikaryotic mycelium
if heteroecious:
host A
host B
I aeciospores
dikaryotic
dikaryotic mycelium
II urediniospores
dikaryotic
REPEATING STAGE
III teliospores
diploid
KARYOGAMY MEIOSIS

Figure 2.33. Heteroecious, macrocyclic rust life cycle with Roman numerals.

Aeciospores (spore stage **I**) are dikaryotic spores produced in **aecia** from the newly formed dikaryotic mycelium. If the rust fungus is **heteroecious**, requiring two unrelated host plants to complete its life cycle, this is the stage that infects the unrelated host species. In heteroecious rusts, aeciospores are unable to infect the plant on which they were produced. Usually, large numbers of aeciospores are produced to help ensure that some are successfully dispersed to the other host species. The germinating aeciospore establishes the dikaryotic mycelium in that host species.

Typically, this mycelium soon produces another kind of dikaryotic spore, **urediniospores** (spore stage **II**) in **uredinia**. These are the powdery, rust-colored spores that give rust diseases their name. Urediniospores are called the **repeating stage** because they are the only rust spores that can cause new infections of the host species on which they were produced.

The survival spores of rust fungi are **teliospores** (spore stage **III**) formed in **telia**. Teliospores are produced from the same dikaryotic mycelium that produced the urediniospores. The dikaryotic nuclei of the teliospore fuse, forming the only diploid stage in the entire life cycle, and undergo meiosis to produce four haploid **basidiospores** (spore stage **IV**). This step provides the major source of genetic variation in rust fungi. The basidiospores infect only the initial host species on which the pycnia are produced, so they must be dispersed back to that host species.

Spores and Their Functions in Heteroecious Rust Fungi

basidiospores (IV)

- are haploid
- infect aecial (alternate) host species to form pycnia

pycniospores (0)

- combine with receptive hyphae in pycnia
- result in formation of dikaryotic mycelium (plasmogamy)

aeciospores (I)

- are dikaryotic spores produced in aecia
- infect telial (primary) host

urediniospores (II)

- are dikaryotic spores produced in uredinia
- infect the host species on which they are produced (repeating stage)

teliospores (III)

- are diploid spores produced in telia (dikaryotic, becoming diploid)
- produce haploid basidiospores (after karyogamy and meiosis)
- serve as the survival stage

Authors of publications on plant diseases may refer to host plants of heteroecious rusts as the **telial** (**primary**) **host** (look for uredinia and telia) or the **aecial** (**alternate**) **host** (look for pycnia and aecia) when they want readers to know which spore stages are on which host species. However, different uses of the term “alternate” in rust disease cycles can lead to confusion. Plant pathologists usually refer to the two hosts required by a heteroecious rust fungus to complete its life cycle as the alternate hosts. Sometimes, the term alternate host is used to designate the host plant of lesser economic value.

Another way to designate the spore stages is with the Roman numerals indicated above. The role of pycniospores was not discovered until 1927, after the original numbers had already been assigned to the other spore stages, so pycniospores were designated stage 0. Authors may use (II, III) next to a host plant name to indicate the telial host and (0, I) to indicate the aecial host. Not all rust fungi are heteroecious; some rusts are **autoecious** and may produce all spore types on one host.

Autoecious rust fungi require a single host species to complete their life cycle (similar to most fungal pathogens).

Heteroecious rust fungi require two unrelated plant species to their complete life cycle.

► DID YOU KNOW?

Anton deBary first studied bean rust and determined the stages of an autoecious, macrocyclic rust fungus. He then turned his attention to the more economically important wheat stem rust. Using the farmers' belief that barberries increased the incidence of wheat rust, he successfully inoculated barberries with basidiospores of *Puccinia graminis* and observed the development of the subsequent spore stages. Once the amazing nature of the heteroecious life cycle was understood, many other rust fungi were found to be heteroecious and their hosts could be paired up.

The life cycles of most **smut fungi** are not as complex as those of the rust fungi, but they differ from those of most basidiomycetes. Each cell of the dikaryotic mycelium forms a dark, thick wall and eventually breaks apart, forming a mass of powdery **teliospores**. This process appears to be similar to the formation of **chlamydospores**, which are asexual survival spores produced by other fungi. Unlike chlamydospores, however, smut teliospores are sexual spores in which the dikaryotic nuclei fuse, undergo meiosis, and produce haploid nuclei. The haploid spores are similar to basidiospores, but they are called **sporidia** in the smuts. Through a variety of mechanisms, depending on the specific smut fungus, these haploid nuclei eventually pair to form the dikaryotic mycelium that is capable of causing a new smut infection.

Oomycota

OOMYCETES

- **nonseptate** hyphae
  - **diploid** nuclei
- cell wall containing cellulose (**no chitin**)
- sexual spores: **oospores**, produced after contact between an oogonium and an antheridium
- asexual spores: **zoospores**, with two **flagella**, produced in a **sporangium**

Nuclei in the hyphae of oomycetes are **diploid**, unlike true fungi, whose hyphae have haploid nuclei. Meiosis occurs in the **oogonium** (female sexual structure) and the **antheridium** (male sexual structure). Antheridia contribute haploid nuclei to those in the oogonia. Following plasmogamy and karyogamy, the diploid state is restored in the **oospore**.

Oomycetes are identified by the number and location of antheridia relative to the oogonium, characteristics of the oospore, and characteristics of the sporangium, in which zoospores are typically produced. Antheridia attached to the side of an oogonium are **paragynous** (Figure 2.34A), which is typical of most *Pythium* species. There may be one or several antheridia. Alternatively, the oogonium may grow through a single antheridium, which is then attached like a collar at the base of the oogonium and called **amphigynous** (“around the female”) (Figure 2.34B). Antheridia of

Phytophthora species may be paragynous or amphigynous, but there is usually only one antheridium per oogonium.

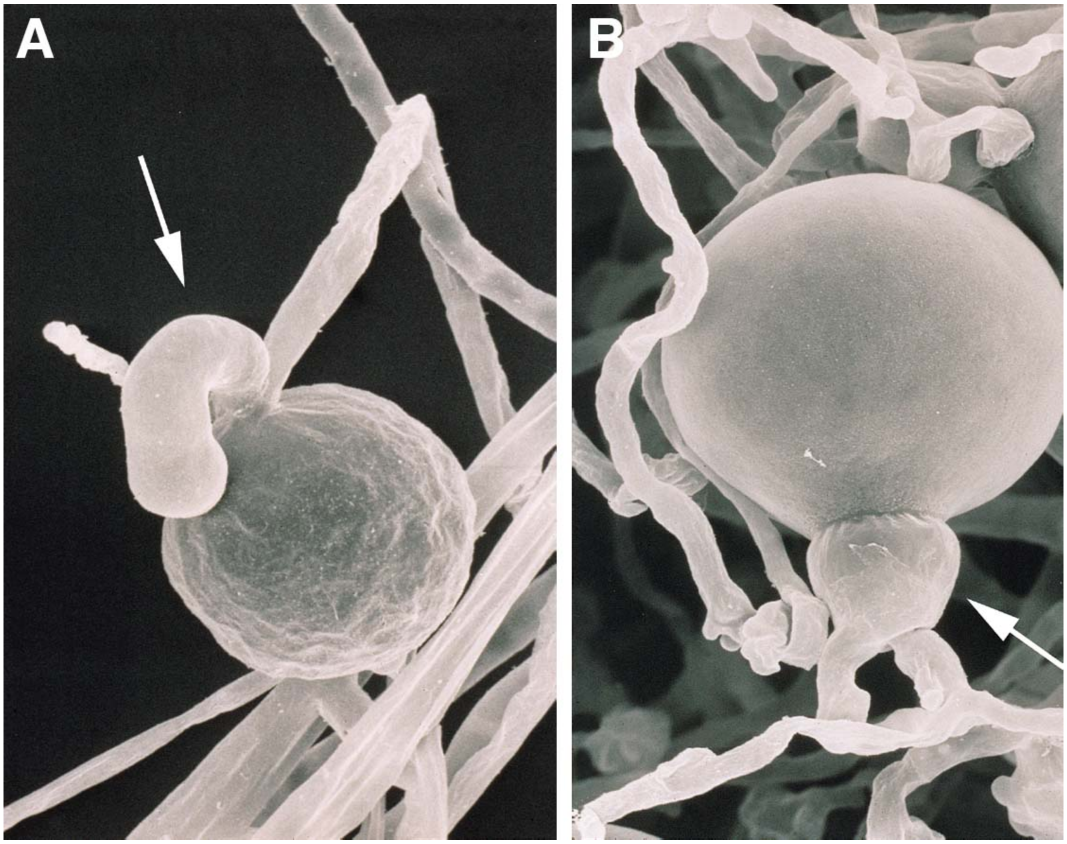


Figure 2.34. A, Paragynous antheridium (arrow) of *Pythium* sp. B, Amphigynous antheridium (arrow) of *Phytophthora* sp.

Zoospores are the asexual stage of oomycetes and are produced in a **sporangium.** In *Pythium* species, zoospores do not form inside the sporangium. The cytoplasm in the sporangium flows into a fragile vesicle where the zoospores quickly form and swim away. The shapes of oomycete sporangia vary from looking like a lumpy hypha to a more defined lemon or round shape.

Important Oomycetes

Pythium species

- **Nonseptate** hyphae

- **Sexual spores**: oospores; paragynous antheridia, often more than one per oogonium
- **Asexual spores**: zoospores released from vesicles produced by lumpy to round sporangia
- **Diseases**: seedling damping-off; stem, crown, and root rots; diseases of lower tissues reached by water splashing up from the soil (the "splash zone")

***Phytophthora* species** (*phyto* = **plant**, *phthora* = **destroyer**)

- **Nonseptate** hyphae
- **Sexual spores**: oospores; paragynous or amphigynous antheridium; usually only one antheridium per oogonium
- **Asexual spores**: zoospores produced by typically lemon-shaped sporangia
- **Diseases**: stem, crown, and root rots; diseases of lower tissues reached by water splashing up from the soil

Phytophthora infestans causes late blight of potato and tomato, the disease that led to the Irish potato famine in the 1840s and the beginning of the science of plant pathology.

Phytophthora ramorum causes the devastating ramorum blight (sudden oak death) on the U.S. west coast and was probably introduced into the United States on rhododendron nursery stock. *Phytophthora cinnamomi* is a widespread, destructive root and crown pathogen throughout the tropical world.

Downy mildew pathogens (biotrophs)

Plasmopara, Peronospora, Pseudoperonospora, Bremia, and other genera

- **Nonseptate** hyphae
- **Sexual spores**: oospores, produced in infected, aboveground plant tissue
- **Asexual spores**: sporangia, formed on treelike sporangiophores, which typically emerge through stomata on

the lower leaf surface; some sporangia produce zoospores, and others germinate directly

- **Diseases**: blue mold of tobacco; crazy top of corn; yellow tuft of turfgrasses; downy mildews of brassicas, curcurbits, grape, hops, onion, snapdragon, and other plants

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Questions

1. You observe a fruiting body with an opening in the top through which spores are released. A fruiting body with this characteristic could be one of several types: ascostroma, conidial stroma, perithecium, pseudothecium, or pycnidium. How do you determine which type of fruiting body you are observing?
2. The *Compendium of Cucurbit Diseases* describes the pathogen that causes gummy stem blight as follows:

> Gummy stem blight is caused by *Didymella bryoniae* (syn. *Mycosphaerella melonis*). Dark pycnidia of the anamorph, *Phoma cucurbitacearum*, 120–180 μm in diameter, form on the surface of leaves, stems, and fruits. The conidia are hyaline, cylindrical with rounded ends, non- or monoseptate, and 6–13 μm long. Dark pseudothecia, 125–213 μm in diameter, may also form, especially on stems. The numerous bitunicate asci usually produce eight ascospores per ascus. The ascospores are 14–18 × 4–6 μm, hyaline, monoseptate with a constriction at the septum, and rounded on the ends. The upper cell is usually wider than the lower cell.

Use your new vocabulary to draw the features of the fungal pathogen that you would look for if you suspected that a cucumber, melon, or squash plant has this disease. To which fungal group does this pathogen belong? Will the hyphae be septate or nonseptate?

Challenge Questions

1. Cucumbers are grown in soil outdoors and in hydroponic (soilless) systems in greenhouses. In which situation would downy mildew be more of a problem and why?
2. *Puccinia graminis*, the wheat stem rust fungus, has five spore stages. *Venturia inaequalis*, the apple scab fungus, has two spore stages. Which spore stages of these fungi have parallel functions? Which rust spores have a unique function for which there is no parallel spore in the apple scab fungus?

Supplementary Resources

Illustrated Glossary

Special Topic

Why are *Phytophthora* and other Oomycota not true fungi?

Plant Disease Profiles

Ascomycetes

Anthracnose of turfgrass
Apple scab
Black knot
Black Sigatoka of bananas and plantains
    Sigatoka negra (Spanish)
    Sigatoka negra (Portuguese)
Blackleg of oilseed rape
Brown rot of stone fruits
Dollar spot of turfgrass
Dutch elm disease

- Early blight of potato and tomato
    - Pinta-preta (Alternariose) da batata e tomate (Portuguese)
- Ergot
    - Cornezuelo del centeno (Spanish)
- Fusarium head blight
    - Giberela ou Fusariose (Portuguese)
- Fusarium wilt of watermelon and other cucurbits
- Leucostoma canker of stone fruits
- Monosporascus root rot and vine decline of melons
- Rice blast
- Sudden death syndrome of soybean
    - Síndrome de la muerte súbita (SMS) de la soja (Spanish)
- Take-all root rot
- Verticillium wilt
- White mold (Sclerotinia)

Basidiomycetes

- Armillaria root disease
- Brown root rot
- Coffee rust
- Common smut of corn
- *Rhizoctonia* diseases of turfgrass
- Southern blight
- Soybean rust
    - Ferrugem asiática da soja, Ferrugem da soja (Portuguese)
- Stem rust of wheat
- Stinking smut of wheat
- White pine blister rust

Oomycetes

- Aphanomyces root rot or common rot of legumes
- Black shank of tobacco
- Downy mildew of grape

- Late blight of potato and tomato
    - Requeima (mildio, pt) da batateira e tomateiro (Portuguese)
    - 马铃薯和番茄晚疫病 (Chinese)
- Phytophthora blight of cucurbits
- Phytophthora root and stem rot of soybean
- Pythium blight of turfgrass
    - Quemazón del césped por Pythium (Spanish)
- Sudden oak death, ramorum leaf blight, ramorum shoot blight
- Taro leaf blight

Laboratory Exercises

- Basic microscopy—An important skill for plant pathologists
    - Microscopía básica—Una destreza importante para fitopatólogos (Spanish)
- Cytology of fungal infection
- Following the disease progression of an ectotrophic root-infecting fungus
- Identification of powdery mildew fungi anno 2006
- Oomycetes

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

- anamorph
- ascocarp
- ascoma (pl. ascomata)
- ascospore
- ascus (pl. asci)
- aseptate
- avoidance

basidiocarp
basidioma (pl. basidiomata)
basidiospore
basidium (pl. basidia)
biotroph
chitin
chlamydospore
clamp connection
coenocytic
conidioma (pl. conidiomata)
conidiophore
conidium (pl. conidia)
dikaryotic
diploid
disinfect
disinfest
downy mildew
eradication
eukaryote
exclusion
flagellum (pl. flagella)
fruiting body
fungus (pl. fungi)
genus (pl. genera)
haploid
heterothallic
heterotroph
holomorph
honeydew
hypha (pl. hyphae)
infect
infest
karyogamy

meiosis
melanin (melanized)
mycelium (pl. mycelia)
mycology
necrotroph
nonseptate
oospore
ostiole
plasmodium (pl. plasmodia)
plasmogamy
protection
rust
saprophyte
sclerotium (pl. sclerotia)
septate
septum (pl. septa)
slime mold
smut
sp. (pl. spp.)
species
specific epithet
sporangiophore
sporangium (pl. sporangia)
spore
teleomorph
teliospore
urediniospore
white rust
zoospore
zygospore

► MORE SPECIALIZED VOCABULARY

acervulus (pl. acervuli)

aecial host
aeciospore
aecium (pl. aecia)
alternate host
amphigynous
antheridium (pl. antheridia)
apothecium (pl. apothecia)
ascostroma (pl. ascostromata)
autoecious
bitunicate
chasmothecium (pl. chasmothecia)
cleistothecium (pl. cleistothecia)
coelomycetes
heteroecious
hyphomycetes
oogonium (pl. oogonia)
paragynous
perithecium (pl. perithecia)
primary host
pseudothecium (pl. pseudothecia)
pycnidium (pl. pycnidia)
pycniospore
pycnium (pl. pycnia)
receptive hypha (pl. receptive hyphae)
seta (pl. setae)
sporidium (pl. sporidia)
sporodochium (pl. sporodochia)
stroma (pl. stromata)
synnema (pl. synnemata)
telial host
teliospore
telium (pl. telia)
unitunicate

uredinium (pl. uredinia)

Exercises

Exercise 2.1. Identify each of the fungal structures or features.

Exercise 2.2. Challenge identification of fungal structures or features.

Internet Resources

APS Resources

Brown Rot of Dried Plum in California: New Insights on an Old Disease
Flora W. Patterson: The first woman mycologist at the USDA
Potato wart
Snow mold-plant-antagonist interactions: Survival of the fittest under the snow
Videoclip of zoospore release and swimming

Recommended Websites

Cornell Mushroom Blog
Cornell Time Lapse Movie Gallery
The Microbial World: The Fungal Web
Tom Volk's Fungi

Internet Research

Internet Research Exercise 2.1. Find a fact sheet or other description of a fungal plant disease. (See Appendix 2 for lists of important diseases of specific plants. See the Supplementary Resources for a list of recommended websites with fact sheets.)

a. What is the name of the pathogen?
b. What are the host plants?
c. What are the symptoms and signs of the disease?
d. What are the features of the mycelium, spores, and fruiting bodies of the pathogen?
e. To what fungal group does this pathogen belong?
f. What environmental conditions favor the disease?

Internet Research Exercise 2.2. Your neighbor calls you in a panic because something large and disgusting has suddenly appeared on his shredded bark mulch after a warm, rainy night in July. He worries that it will attack his lawn and roses. Read about the famous dog vomit slime mold, *Fuligo septica* (myxomycete), and give him a recommendation.

See Tom Volk's Fungi (website), Fungus of the Month, June 1999

CHAPTER 3

What Are the Causes of Plant Diseases?

► Bacteria

IN THIS CHAPTER:

- ► **What are bacteria?**
- ► **What symptoms and signs do bacteria cause?**
- ► **How do bacteria survive and spread?**
- ► **How are bacterial diseases diagnosed?**
- ► **How are bacterial diseases managed?**

What are bacteria?

Almost all bacteria (Figure 3.1) are tiny (1–3 micrometers), single-celled organisms, much smaller than fungal spores. **Bacteria** (singular: bacterium) are **prokaryotic** (pro = before, karyo = nucleus) organisms, which means that they do not have membrane-bound nuclei or other organelles, such as mitochondria. Most bacteria have **cell walls**, which determine the shape of the bacterium: round (**coccal**), spiral-shaped, or rod-shaped (**bacilliform**) (Figure 3.2). Nearly all plant-pathogenic bacteria are bacilliform. Some bacteria lack a cell wall, however, and their shapes can change. These organisms, called **mollicutes**, include **phytoplasmas** (which are **pleomorphic**, or variably shaped)

(Figure 3.3) and **spiroplasmas** (which are usually helical) (Figure 3.4). Finally, some bacteria produce long, branched filaments. These soil inhabitants, called **actinomycetes**, are known to produce inhibitory compounds (**antibiotics**) that affect the growth and development of other organisms.

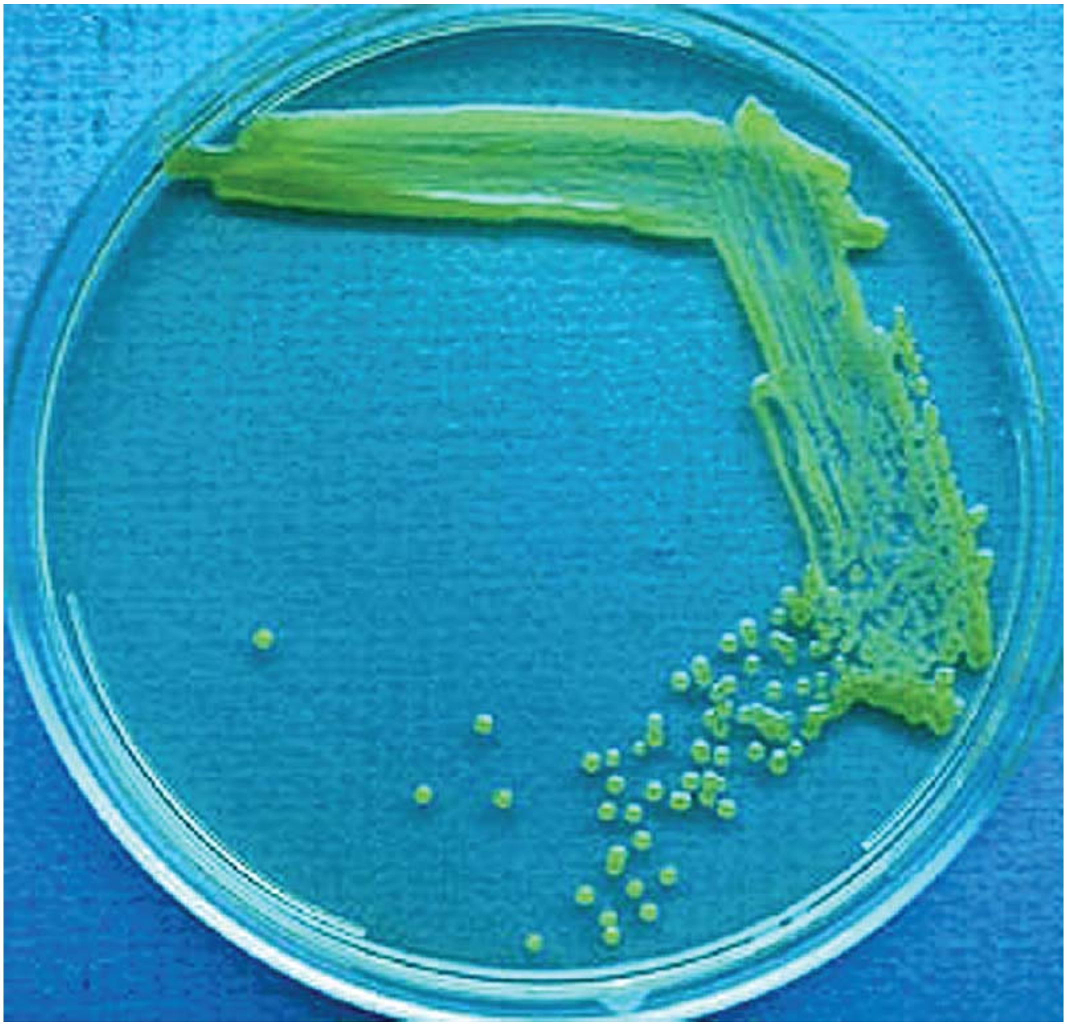

Figure 3.1. Bacterial colonies on a nutrient medium. Note that the bacteria have been diluted through streaking. The small, round colonies are made up of thousands to millions of cells, and each probably grew from a single bacterium.

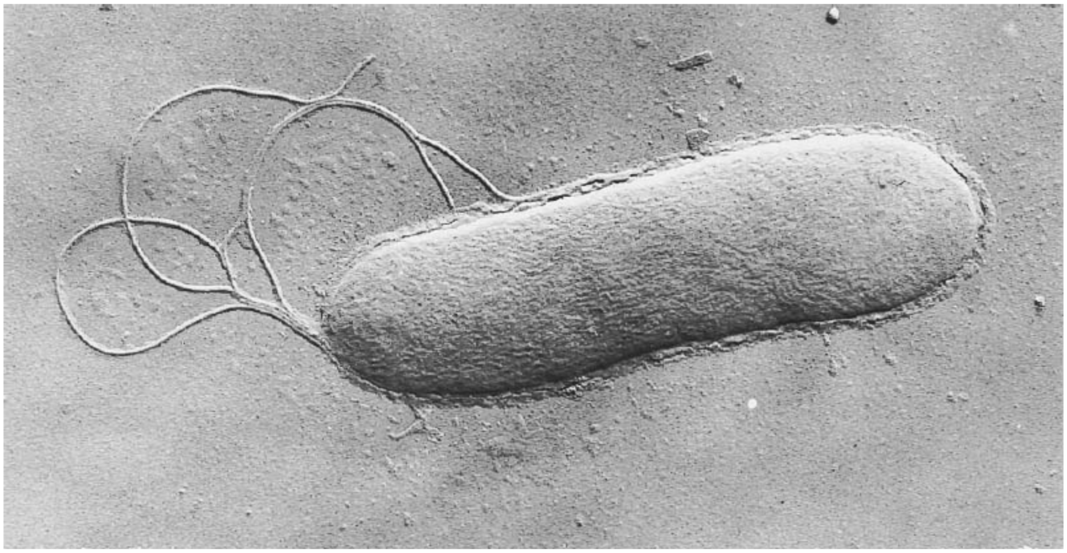

Figure 3.2. A bacilliform bacterium, typical of plant pathogens. Note that it has polar flagella.

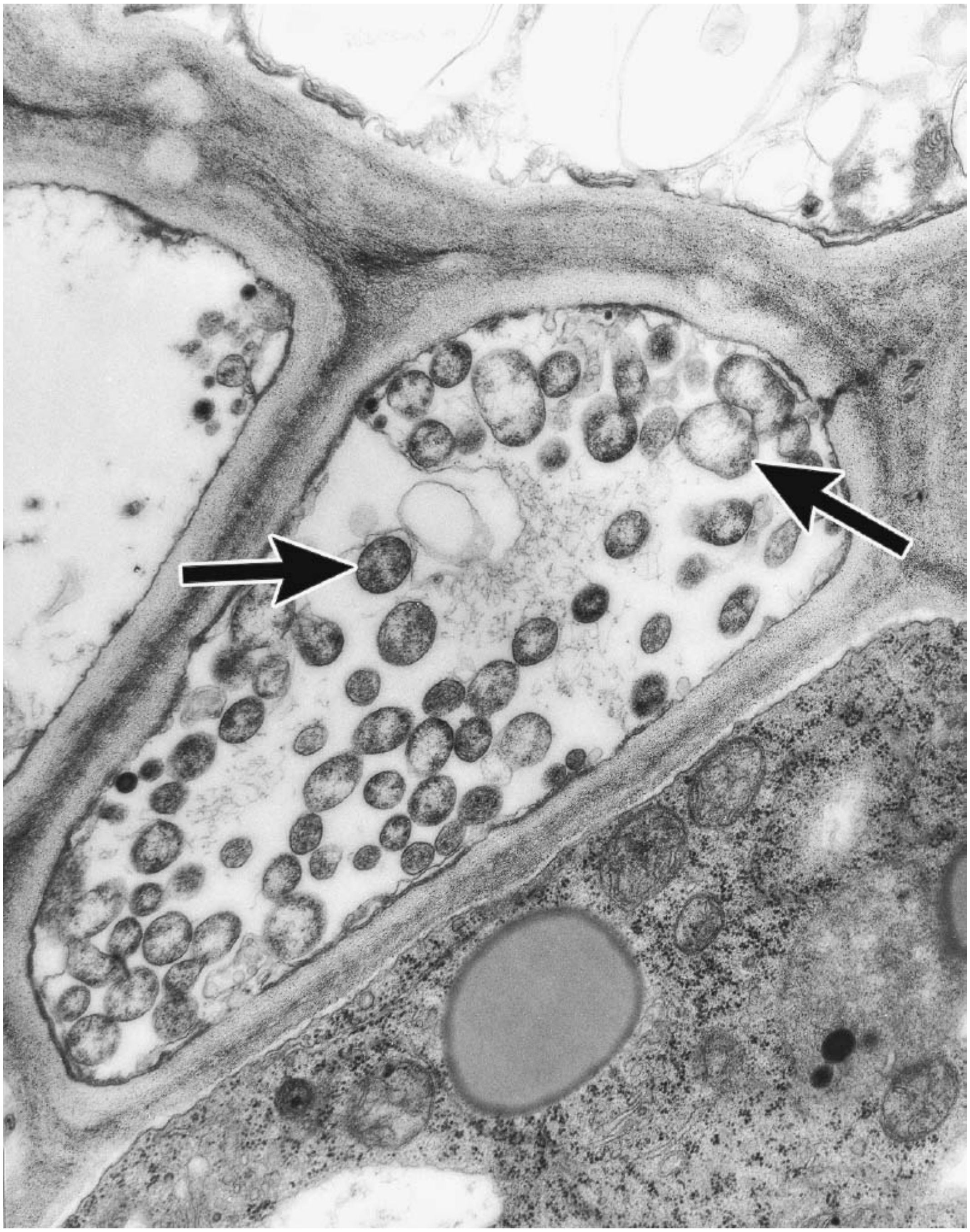

Figure 3.3. Phytoplasmas (arrows) in a phloem cell of an infected plant. Note that the phytoplasmas are pleomorphic (variably shaped).

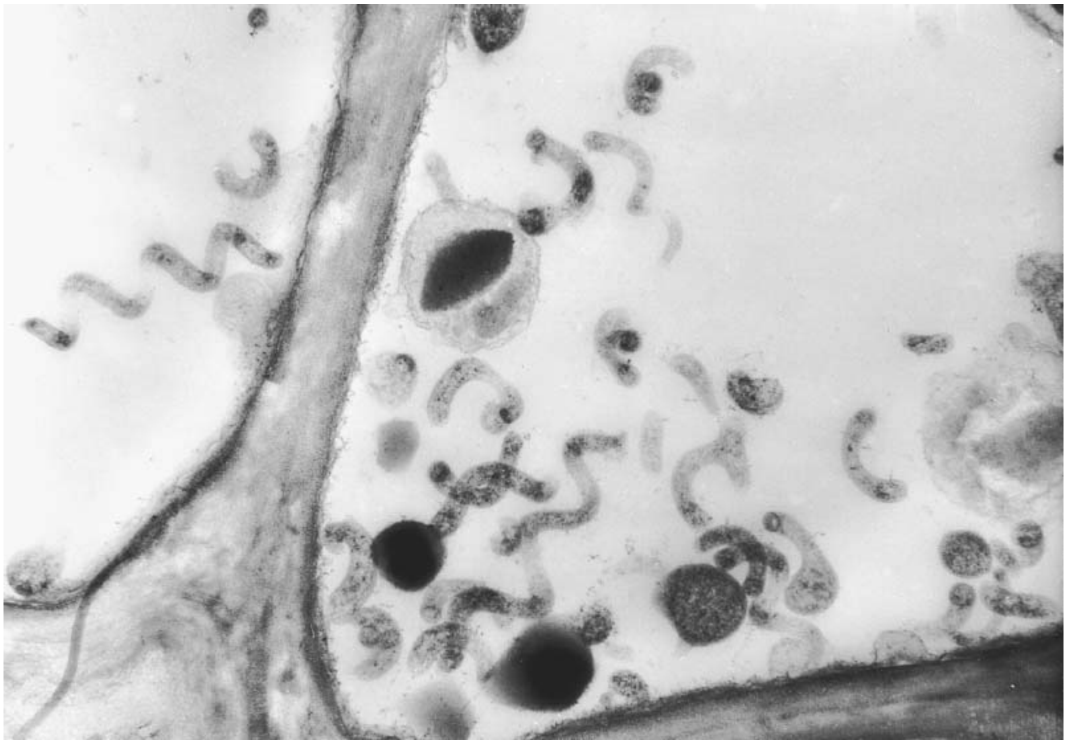

Figure 3.4. Spiroplasmas in phloem cells of an infected corn plant. Note the helical shape.

Many bacteria secrete **extracellular polysaccharides** (EPS), high molecular weight carbohydrates that become attached to the exterior surface of the cells as a slime layer (**capsule**) (Figure 3.5) and serve to promote the disease process. Bacteria also often have **flagella**, external structures that rotate to propel the cells short distances through liquids. The location of flagella, either at the ends of the bacterial cells (**polar**) or all over the cell surface (**peritrichous**), is a useful taxonomic characteristic. Polar flagella are evident in Figure 3.2.

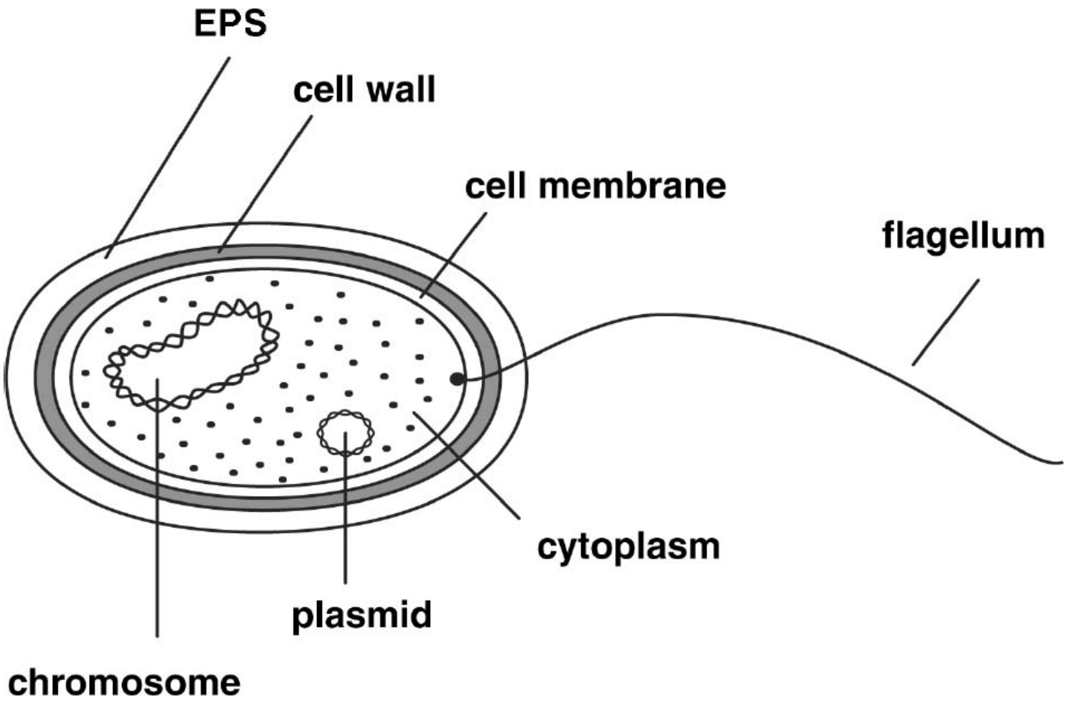

Figure 3.5. Bacterial cell. Note the EPS (extracellular polysaccharide) and polar flagellum.

Bacterial cells contain small **ribosomes** (involved in protein synthesis) and a haploid DNA **genome** that consists of a single chromosome tangled within an area called a **nucleoid**. The genetic code of bacteria is nearly identical to that of higher organisms. In addition to the chromosome, their cells may contain small, circular pieces of DNA called **plasmids**. Plasmids replicate independently of the chromosomal DNA and may code for proteins important in the disease development process.

Bacteria reproduce by a process called **fission**, in which a single cell divides into two identical cells following the replication of the chromosomal DNA. Although bacteria do not undergo true sexual reproduction, they have several means of genetic change, including the exchange of genetic information between bacterial cells. Part or all of the chromosome can be directly transferred from one bacterium to another during **conjugation**, and pieces of the genome can be transferred by plasmids or by **bacteriophages** (bacteria-infecting viruses). The most common source of genetic change in bacteria is **mutation**, mistakes that occur during replication of the DNA. Under optimal conditions, some bacteria can divide every 20 minutes. Since their DNA is replicated during each fission cycle, there are frequent opportunities for mutation.

Most plant-pathogenic bacteria are **facultative parasites** that readily adapt to a variety of environments. As a result, it is relatively easy to culture them on standard nutrient media. Individual bacterial cells are not visible with the naked eye, but each bacterium divides many times and forms a small, round **colony** of cells that is visible on the agar surface. Some bacteria that infect plants have never been cultured or can be cultured only on complex, specialized media, leading to their designation as **fastidious bacteria**. Some fastidious bacteria have cell walls (e.g., **xylem-limited bacteria**), whereas others are the wall-less **phytoplasmas** and **spiroplasmas**.

Many plant-pathogenic bacteria exist as **epiphytes** on plant surfaces, living on exudates from plant cells, and become pathogens only when conditions are conducive for infection. Moist, warm conditions usually favor bacterial growth and colonization of plant tissues. Most plant-pathogenic bacteria are **aerobic** (requiring oxygen to live), although a few are **facultative anaerobes** that grow best with oxygen but can live without it for a while.

► DID YOU KNOW?

In 1878, Thomas J. Burrill (Figure 3.6) (1839–1916), who taught at the University of Illinois, was the first scientist to propose that bacteria can cause plant diseases. In his studies on fire blight (caused by *Erwinia amylovora*), he found that “…under our microscope, the field is seen to be alive with moving atoms known in a general way as bacteria.” Burrill’s proposal was later proved correct when Koch’s postulates were fulfilled by J. C. Arthur in his Ph.D. thesis work at Cornell University.

Figure 3.6. Thomas J. Burrill in a laboratory with students, 1882.

There are thousands of named species of bacteria, but only about 30 genera include plant pathogens, which cause hundreds of plant diseases. These genera can be divided into two major groups based on the structure of the bacterial cell wall, using the **Gram stain**. One group of bacteria, with thick cell walls, traps a dye and becomes purple (called **gram-positive**). The other group has thin cell walls that do not retain the purple dye (**gram-negative**). A different stain must be used to make the gram-negative bacteria visible, commonly basic fuchsin or safranin, which stains them pink. Most plant-pathogenic bacteria are gram-negative. More than 300 known diseases of plants are caused by phytoplasmas, but only a few are known to be caused by spiroplasmas.

Common Plant-Pathogenic Bacterial Genera: Characteristics and Examples

Gram-negative

Agrobacterium

- aerobes, peritrichous flagella, abundant EPS
- Example: *Agrobacterium tumefaciens*

Erwinia and closely related newer genera: ***Brenneria, Pantoea, Pectobacterium***

- facultative anaerobes, peritrichous flagella; pectolytic enzymes (some species)
- Examples: *Erwinia amylovora* (no pectolytic enzymes), *Pectobacterium carotovorum* (pectolytic enzymes)

Pseudomonas and related newer genera: ***Acidovorax, Burkholderia, Herbaspirillum, Ralstonia***

- aerobes, polar flagella; some fluoresce under ultraviolet light and chelate (remove) iron from their environment
- Example: *Pseudomonas syringae* (many pathovars)

Xanthomonas

- aerobes, one polar flagellum, yellow colonies; source of “xanthan gums”
- Examples: *Xanthomonas axonopodis* pv. *citri, X. axonopodis* pv. *vesicatoria*

Gram-positive

Clavibacter

- aerobes, irregularly shaped rods, nonmotile
- Examples: *Clavibacter michiganensis* subsp. *michiganensis*, *C. michiganensis* subsp. *sepedonicus*

Streptomyces

- aerobes, branched filaments, spore-forming (actinomycete)
- Example: *Streptomyces scabies*

► **DID YOU KNOW?**

Phytoplasmas were once called “mycoplasma-like organisms” or MLOs. Mycoplasmas were first discovered as animal and human pathogens. When similar organisms were discovered in the phloem cells of plants but could not be cultured, they were called MLOs. Later, when molecular tools showed that the wall-less, pleomorphic bacteria in plants were distinct from animal mycoplasmas, they were renamed “phytoplasmas.”

Usually, bacteria can be identified to genus using a series of standard laboratory **selective media** and physiological tests. Identification to species is more difficult and is often done using immunoassays and DNA-based tests such as the polymerase chain reaction (PCR) (see Appendix 3).

The scientific names of bacteria are Latin binomials (genus and specific epithet), which are used also for animals, fungi, nematodes, and plants. Some plant-pathogenic bacteria, however, have a third part to their name, the **pathovar**, which is abbreviated **pv**. This term is used to differentiate bacteria that appear to belong to the same species but infect different host species. For example, *Xanthomonas axonopodis* pv. *citri* causes citrus canker, whereas *X. axonopodis* pv. *vesicatoria* causes bacterial spot of peppers and tomatoes. Some other bacterial names include the taxonomic rank of **subspecies** to separate strains that can be distinguished by features other than pathogenicity, including morphological, physiological, or genetic differences. For example, *Clavibacter michiganensis* subsp. *sepedonicus* causes ring rot of potato, whereas *C. michiganensis* subsp. *michiganensis* causes bacterial canker of tomato.

► **DID YOU KNOW?**

Xanthan gums are extracellular polysaccharides produced by *Xanthomonas* bacteria as protective substances and an external food source. They also help the bacteria stick to the leaves of host plants. The gums are produced commercially in industrial-sized tanks and used for a variety of purposes such as the food thickeners commonly found in dipping sauces, salad dressings, and chewing gum.

Bacteria are small, single-celled prokaryotic organisms that reproduce by binary fission and evolve mainly through mutation. Most plant-pathogenic bacteria have cell walls and are bacilliform, but some, called mollicutes, lack cell walls and assume other shapes. Bacteria are classified into about 30 different genera, most of which are gram-negative.

What symptoms and signs do bacteria cause?

Bacteria cause many of the same kinds of diseases in plants that fungi do: cankers, galls, leaf spots, rots, and vascular wilts. These are good examples of why symptoms alone often are not sufficient for an accurate diagnosis. For many bacterial diseases, the number of bacteria in infected tissue is enormous. Sometimes enough bacteria accumulate to produce a sign called **ooze** (Figure 3.7), which is a sticky liquid composed of extracellular polysaccharides (EPS) and bacterial cells. Ooze can be exuded from natural plant openings or from the edges of cankers, as in fire blight (*Erwinia amylovora*). In cucurbits with bacterial wilt (*Erwinia tracheiphila*), strands of ooze can be seen when a cut stem is slowly drawn apart.

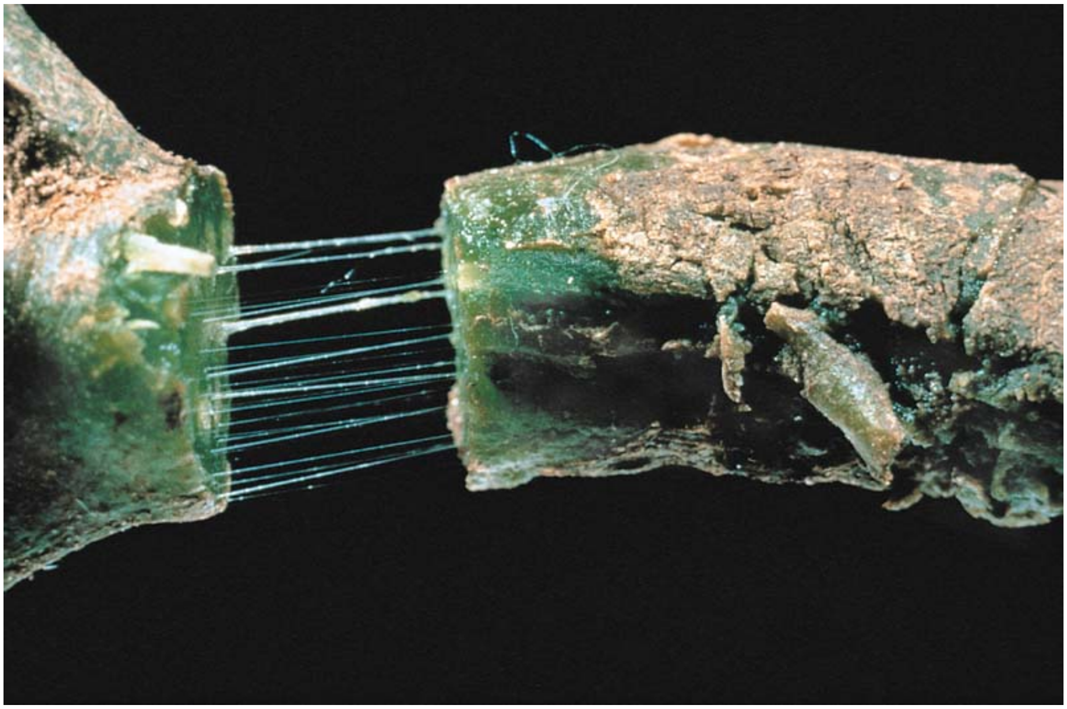

Figure 3.7. Ooze, a diagnostic sign of a bacterial infection. In bacterial wilt of cucurbits, the sticky strands of bacterial ooze become evident as two portions of a cut stem are pulled apart.

Another sign in some bacterial diseases is **bacterial streaming** (Figure 3.8). Under a microscope, millions of bacteria can be seen streaming out of leaf blades cut from plants with a foliar disease or stems cut from plants with a bacterial wilt. Bacterial streaming can be misleading, however, because saprophytic bacteria quickly colonize dead and dying tissues caused by another primary agent. Therefore, bacterial streaming is most significant when observed in the early stages of a disease.

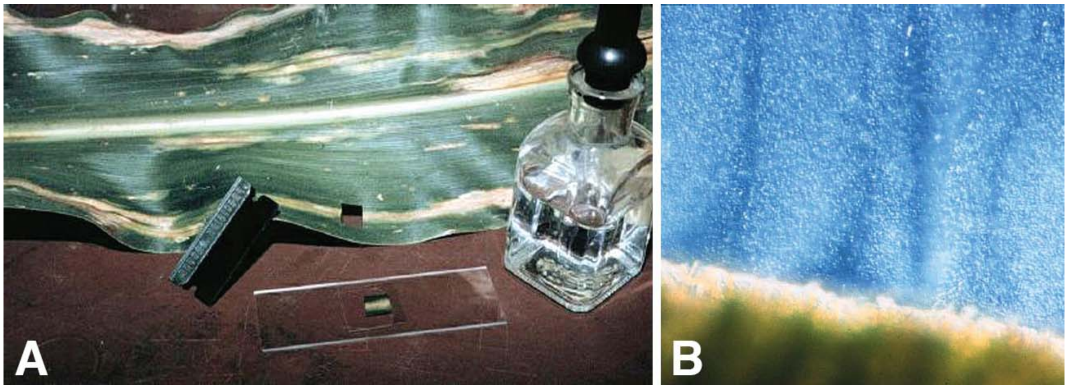

Figure 3.8. A, To diagnose Stewart's wilt, a section of symptomatic corn leaf tissue is placed in a drop of water on a microscope slide. B, Bacterial streaming, viewed with a compound microscope.

Leaf symptoms can suggest whether a disease is caused by a bacterium or a fungus. As discussed previously, fungi tend to cause round leaf spots as the mycelium grows out radially from the infection point. Bacteria usually move **intercellularly** (between cells) through leaf tissue and can be more easily blocked by leaf veins that would not inhibit a fungus. Thus, bacterial leaf spots tend to be more angular or irregular in shape, depending on the anatomy of the leaf. Further investigation may be required to determine whether bacteria are the cause of a lesion, because leaf spots can be quite variable.

Bacteria often cause **water-soaking** of the host tissue due to enzymatic destruction of the plant cells, or they may cause a **yellow halo** of dying cells at the border of the leaf spot due to bacterial **toxins** (poisons) that are present in high enough concentration to damage the plant. Halos also can be seen with fungal infections, but when seen along with the **angular spot** symptom (Figure 3.9), they are most likely to be caused by a bacterial pathogen. Bacterial streaming and the absence of fungal signs can help provide evidence that the disease is caused by a bacterium.

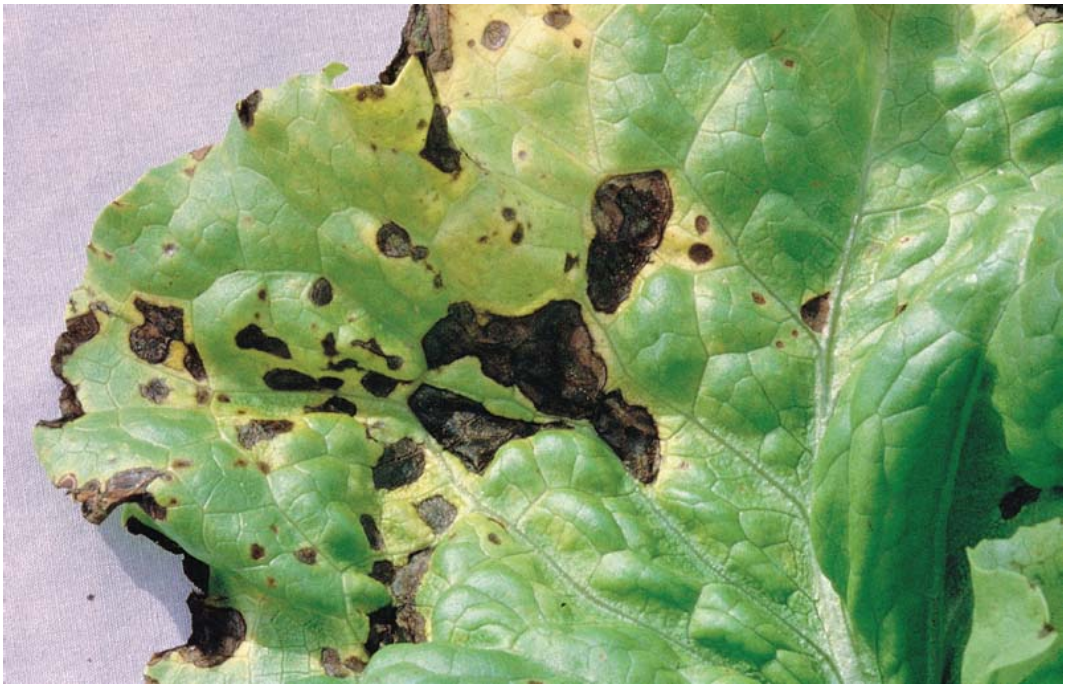

Figure 3.9. Angular leaf spots typical of those caused by *Pseudomonas* spp. and *Xanthomonas* spp.

Some plant-pathogenic bacteria cause plant tissue to liquefy and collapse, causing a **soft rot** (Figure 3.10). These bacteria produce pectolytic enzymes that can degrade the **middle lamella**, the layer of pectin that cements the cells together. The loss of cellular integrity leads to tissue collapse and also provides the bacteria with nutrients, permitting them to spread through the plant tissue. These bacteria produce additional enzymes that degrade various components of the primary cell wall, eventually leading to cell death.

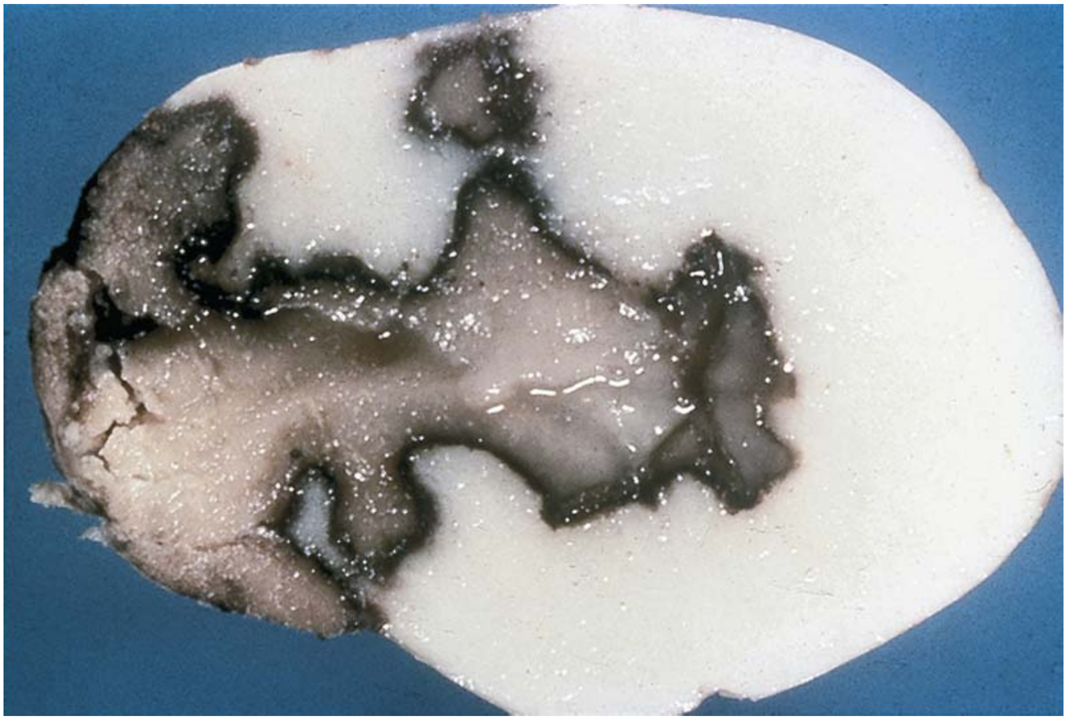

Figure 3.10. Soft rot in a potato tuber.

Agrobacterium tumefaciens is a plant-pathogenic bacterium that induces **galls**, often located near the soil line at the root crown of the infected plant (Figure 3.11). The galls are the result of abnormal plant cell proliferations induced by chemical signals from the bacteria. The bacteria live within the galls, between the plant cells. The crown gall bacterium is famous because it has been used as a "natural genetic engineer" to produce transgenic plants (see Chapters 9 and 11).

Legume nodule bacteria

Nitrogen-fixing bacteria (e.g., *Rhizobium* and *Bradyrhizobium*) associated with legumes, such as soybean and alfalfa, are related to the crown gall bacterium and cause the formation of small galls or "nodules" on host roots. Although dependent on the plant for food, these bacteria are desirable because of their ability to "fix" atmospheric nitrogen into a form that the plant can absorb.

Strains vary in their host plant preferences and in their efficiency as nitrogen-fixers. Legume seeds are inoculated with selected strains to reduce the need for nitrogen fertilizers.

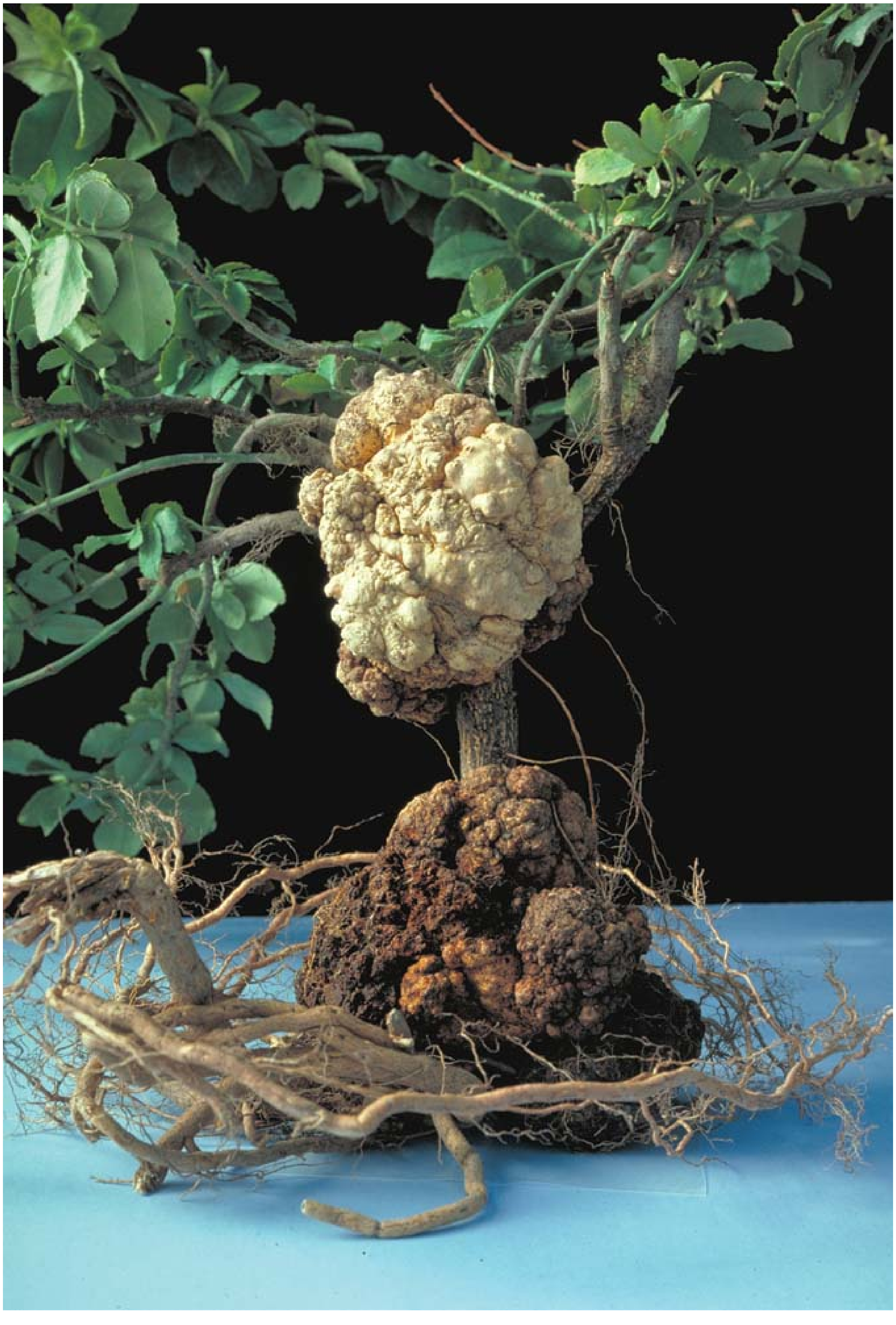

Figure 3.11. Crown gall on euonymous caused by *Agrobacterium tumefaciens*.

Fastidious bacteria usually do not produce obvious signs in infected plants because the bacteria do not accumulate in large enough numbers to produce ooze or bacterial streaming. The symptoms they produce depend on their location in the plant. Bacteria that invade the xylem, including the fastidious *Xylella*, as well as species of *Clavibacter, Erwinia, Pseudomonas, Ralstonia*, and *Xanthomonas*, affect the transport of water and minerals, so **wilting** is a common symptom (Figure 3.12). Wilting also may be due to the accumulation of bacterial EPS, which blocks the flow of xylem sap. Plants in which the xylem is compromised also may be yellowed and stunted, and leaves often show symptoms of **scorch**. Bacteria that invade the phloem, including phloem-colonizing walled bacteria (such as *Serratia* and *Liberobacter*) and mollicutes (phytoplasmas and spiroplasmas) usually induce **yellows** symptoms in leaves, and plants are often stunted. Many of these diseases were originally thought to be caused by viruses because yellowing and stunting are common symptoms of virus infection. Infected plants may exhibit **phloem necrosis** when a stem is cut and also may exhibit various tissue malformations, such as **witches' brooms**, caused by the disruption of the normal balance of growth regulators in the plant (Figure 3.13).

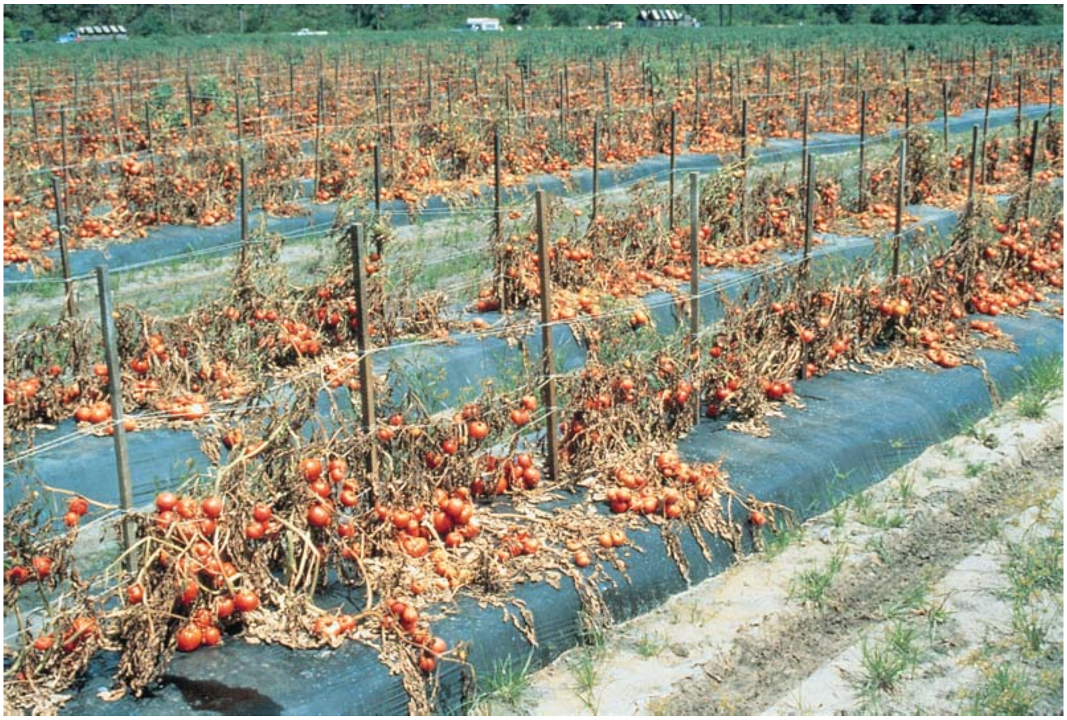

Figure 3.12. Bacterial wilt of tomatoes caused by *Ralstonia solanacearum*.

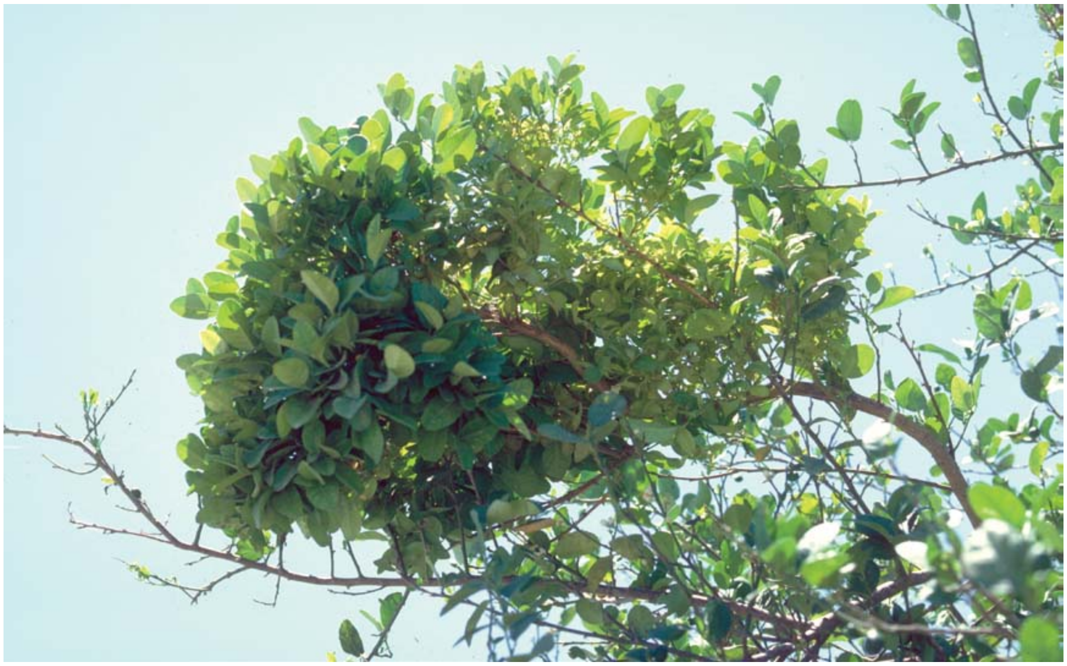

Figure 3.13. Witches' brooming in lime infected by a phytoplasma.

Individual bacteria are too small to be seen with the naked eye, but ooze and bacterial streaming are common signs of

infection. Symptoms include angular spots and blights, soft rots, wilts and, less frequently, gall formation. Fastidious bacteria often cause wilting, yellowing, or malformations.

How do bacteria survive and spread?

Most plant-pathogenic bacteria do not produce special survival structures analogous to the spores produced by fungi, so their ability to survive adverse conditions without a host is limited. Desiccation and damage from ultraviolet radiation are two dangers that bacteria face when they are not protected by a host plant or an insect vector.

Some plant-pathogenic bacteria can survive for a while as **epiphytes** on plant surfaces such as leaves, roots, flowers, and fruits, absorbing nutrients that leak from the plant cells. Bacteria that are thickly coated with EPS may survive longer in this environment because the sticky EPS help retain liquids and keep the bacteria attached to the plant surface. Other plant-pathogenic bacteria are soil inhabitants that can survive for months as **saprophytes**. Only a few plant-pathogenic bacteria survive well in soil, unlike many fungi. Other plant-pathogenic bacteria, including the fastidious bacteria, have no stage of their life cycle that is separate from a plant host or insect vector. These bacteria are transmitted from plant to plant by insects, which is another reason they were originally confused with viruses.

Most plant-pathogenic bacteria survive in association with living tissues, but some can survive in soil or on plant surfaces. Most leaf-spotting bacterial pathogens can survive in dead host residues for short periods. Bacteria invade plants through natural openings or wounds. Inside the host, most bacteria live and move in the intercellular spaces,

although some bacteria invade cells of the phloem or the xylem.

Insect transmission of plantipathogenic bacteria varies depending on the specific pathogen. Many bacteria can be transmitted in a nonspecific way when sticky bacteria contaminate an insect. Sites of insect feeding and egg laying on plants provide wounds for bacterial infection. In other cases, specific vectors have been identified for certain bacterial diseases. Corn flea beetles are the overwintering site and the vectors of the bacterium that causes Stewart’s wilt of corn, and cucumber beetles transmit the bacterium that causes cucurbit bacterial wilt.

The fastidious bacteria are transmitted mostly by insects with piercing-sucking mouthparts (stylets). *Xylella fastidiosa*, a bacilliform bacterium, is transmitted by sharpshooters (Figure 3.14) and multiplies in the food canal of the stylets of these vectors (**propagative transmission**). Mollicutes are transmitted by leafhoppers, and they also multiply in their vectors. For mollicutes, there is a **latent period** of several weeks after the vector acquires them from an infected plant and before they can be transmitted to a new plant, during which time they pass through and multiply within the insect’s body. Mollicutes are sometimes harmful to their leafhopper vectors, leading scientists to hypothesize that mollicutes may have originated as insect pathogens that inadvertently invaded plant phloem during feeding and were able to survive in that protected environment.

Fastidious Vascular-Colonizing Plant-Pathogenic Bacteria

Xylem-limited bacteria

- **Symptoms**: water-stress symptoms including wilt, stunting, scorch on leaves
- **Pathogen**: *Xylella fastidiosa*
- **Shape**: bacilliform (rod-shaped)
- **Vectors**: insects that feed in xylem, e.g., sharpshooters and spittlebugs
- **Example diseases**: bacterial leaf scorch of shade trees, citrus variegated chlorosis, Pierce's disease of grapevine

Phloem-limited bacteria

- **Symptoms**: yellowing, stunting, witches' brooms

Phytoplasmas

- **Pathogens**: no official names yet; *Phytoplasma* genus proposed
- **Shape**: mollicute (no cell wall); pleomorphic (variable shape)
- **Vectors**: leafhoppers
- **Example diseases**: aster yellows, lethal yellowing of coconut, X disease of cherry

Spiroplasmas

- **Pathogens**: *Spiroplasma citri, S. kunkelii*
- **Shape**: mollicute (no cell wall); helical shape
- **Vectors**: leafhoppers
- **Example diseases**: brittle root of horseradish, citrus stubborn, corn stunt, periwinkle spiroplasmosis

Phloem-colonizing walled bacteria

- **Pathogens**: *Serratia marcescens, Liberobacter* spp.
- **Shape**: bacilliform (rod-shaped)
- **Vectors**: psyllids, leafhoppers, one true bug
- **Example diseases**: Huanglongbing (citrus greening), cucurbit yellow vine disease

► DID YOU KNOW?

Some bacteria, such as *Xylella fastidiosa*, find strength in numbers and form “slime communities” called biofilms. Biofilms are populations of bacteria that adhere to environmental surfaces, including plant leaves, stems, and roots. They are found on every surface on which sufficient moisture is present, including industrial equipment, river rocks, and even your teeth! Scientists are now recognizing the importance of biofilms in infectious diseases of both people and plants.

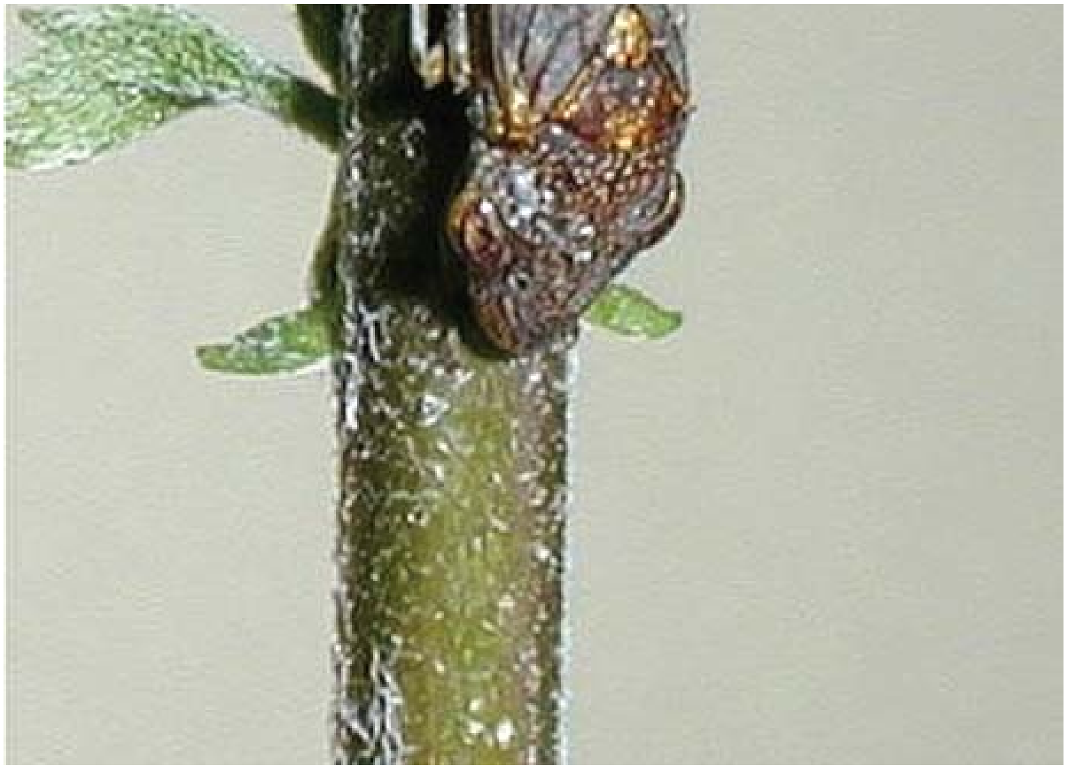

Figure 3.14. The glassy-winged sharpshooter, a leafhopper vector of *Xylella fastidiosa*. These insects subsist on xylem fluid and often feed upside down.

Xylem-Limited Fastidious Bacterium

Although cultured and named as recently as 1978, *Xylella fastidiosa* is the most common xylem-limited pathogen. Its entire genome was sequenced in Brazil, where it causes citrus variegated chlorosis. Brazil produces one-third of the world's oranges and half of the orange juice, so the disease is of great concern there. *X. fastidiosa* also causes a severe scorch disease of many landscape hardwood trees in the mid-Atlantic states. The pathogen was first identified and cultured in the study of Pierce's disease of grapevine. Although this disease has been a problem in California for decades, the 1989 introduction of a new insect vector, the glassy-winged sharpshooter, has threatened the wine industry of the entire state. This vector feeds on more than 133 host plants.

Grape plants with Pierce’s disease must be eradicated to prevent spread. Studies of the gene sequence of *X. fastidiosa* have already helped to explain how the bacterium obtains food from xylem fluid and xylem cells and how it adheres to xylem cells and to the mouthparts of vectors. It is hoped that new management strategies also will be identified.

Plant-pathogenic bacteria, with the exception of those transmitted by specific insect vectors, generally locate a plant host by chance. Although most have flagella, their self-mobility in water is restricted to very short distances. They can respond to signals in their environment, such as nutrients that exude from a host root, and can move along nutrient gradients (**chemotaxis**). However, most plant-pathogenic bacteria spread from host to host by passive movement in flowing and wind-blown water or as contaminants on insects, birds, or people and their tools. Survival of these bacteria therefore depends on the production of a large number of offspring, a few of which will find another host to infect.

Bacteria enter their hosts through natural openings such as **stomata, lenticels**, or **hydathodes** (Figure 3.15). Without such entry points, the surface of a plant leaf or root represents a formidable barrier that a bacterium cannot penetrate by mechanical pressure, as some fungi and nematodes can. Therefore, foliar bacterial infections often increase after rains wash bacteria into these natural openings. For example, the fire blight bacterium, *Erwinia amylovora*, invades flowers through **nectaries**, glands at the base of the flowers. Bacteria are deposited by insect vectors as they feed on and pollinate the flowers.

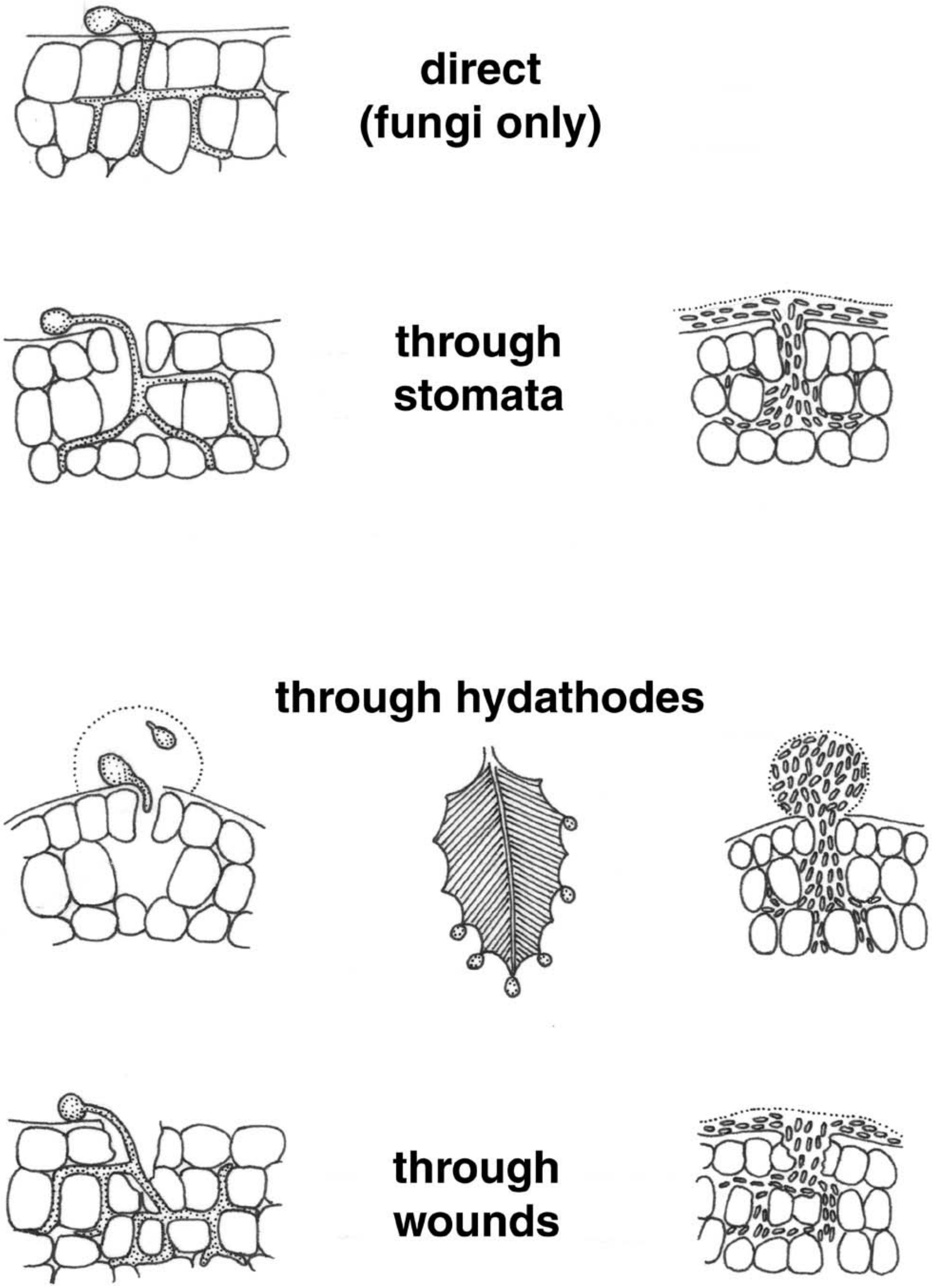
direct
(fungi only)
through
stomata
through hydathodes
through
wounds

Figure 3.15. Various modes of penetration and invasion of a leaf by fungi (left) and bacteria (right). Water is required for bacteria to enter cells.

Wounds are another common entry point for bacteria, and the incidence of most bacterial diseases increases when plants are wounded. Some bacteria, such as *Agrobacterium tumefaciens*, enter plants almost exclusively through wounds. These can be caused by natural plant developmental processes, such as leaf abscission or emergence of lateral root growth, by abrasion caused by wind and blowing soil particles, or by the same vectors that transmit bacteria, such as insects, birds, and people. Bacteria may use lesions caused by other pathogens as entry points. For example, the soft rot bacterium *Pectobacterium carotovorum* can invade potatoes in storage through lesions on the tubers caused by the late blight pathogen, *Phytophthora infestans* (see Chapter 1).

Inside a host plant, bacteria cannot grow through cells as fungi do and are too large to move between cells through plasmodesmata (cytoplasmic strands that connect living cells) as viruses do. Therefore, most bacteria live and multiply in the **intercellular** spaces in leaves, stems, flowers, or galls. Bacterial enzymes and toxins can kill and degrade plant cells or middle lamellae, making cellular products available as nutrients and shutting down plant defenses. Eventually, the bacteria colonize dead host cells, at least until saprophytes move in. In contrast, the fastidious bacteria live **intracellularly** (inside cells), either in the dead cells of the xylem or in the living cells of the phloem. The insect vectors of these bacteria transmit them directly into these plant cells as they feed.

How are bacterial diseases diagnosed?

Diagnosis of common bacterial diseases often requires knowledge of the host plant, the symptoms of the disease, and the genus of the bacterium. For identification to genus, bacteria usually must be isolated in a pure culture, although identification methods that do not require culturing are available (see below). Unlike fungi, which grow radially outward from a piece of infected plant tissue on a culture plate, bacteria multiply to form a sticky puddle surrounding the plant tissue. In this puddle of bacteria is the pathogen, along with any contaminating saprophytes or secondary invaders. It is necessary to separate the primary pathogen from the contaminants, which is done by diluting the bacteria until separate, pure colonies grow from individual bacterial cells on the medium. There are several standard methods to accomplish this. Sometimes repeated streaking of bacteria (**dilution streaking**) (Figure 3.1) on the surface of a nutrient medium with a sterile metal loop is sufficient.

More commonly, infected plant material is chopped up in water to release the bacteria, which are then diluted in sterilized water. Small samples of the water are spread on the medium surface of several petri dishes to find which dilution will produce individual colonies (**dilution plating**) (Figure 3.16). The colony type present in the greatest number is presumed to contain the pathogen. The only way to be certain that the chosen colony type actually contains the pathogen is to inoculate a healthy plant to recreate the same symptoms originally observed on the diseased plant and to then reisolate the same pathogen, in accordance with Koch's postulates (see Chapter 1).

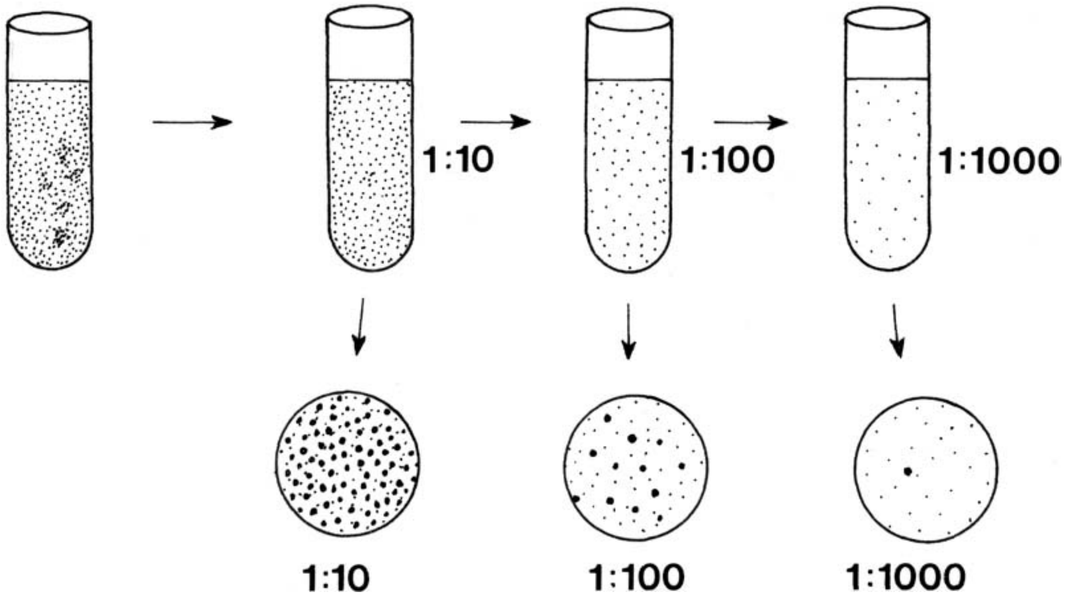

Figure 3.16. Dilution plating to obtain pure colonies of a bacterium. The tissue is torn apart to release the bacteria. The sample is then diluted to

reduce background contaminants. A sample of each dilution is spread on a plate of nutrient medium.

► DID YOU KNOW?

The petri dish was invented by J. R. Petri, a colleague of Robert Koch (of Koch's postulates fame). Petri also invented new methods of culturing bacteria on gelatin. By using shallow, glass dishes with a slightly larger diameter for lids, he made the gelatin in the bottom plate accessible but protected from contamination.

Once the bacterium is in pure culture, standard identification methods can be used. A Gram stain is usually the first step. Gram-positive bacteria are purple when magnified; gram-negative bacteria do not retain the purple color and require another stain to make them visible. Most plant pathogens are gram-negative.

► DID YOU KNOW?

The Gram stain, one of the most important staining techniques in microbiology, was discovered by accident. In 1844, Danish bacteriologist Hans Christian Gram was looking for a stain that would make all bacteria more visible under the light microscope. He discovered that a crystal violet-iodine complex is retained by some bacteria (which appear purple-brown) but not by others. We now know that Gram stain results depend on the structure of the cell wall and can be used to assign bacteria to one of two different biological groups. The Gram stain is almost always the first test performed for the identification of bacteria.

Sometimes the bacteria are examined to determine the number and location of flagella, but this requires special stains and microscopes. For example, *Erwinia* species often have flagella all over their surface (peritrichous flagella) and *Xanthomonas* species have a single flagellum at one end of the rod-shaped cell (polar flagellum).

► **DID YOU KNOW?**

Erwin Frink Smith (1854–1927) was an early bacteriologist who contributed greatly to our knowledge of plantipathogenic bacteria. He added the fourth step to Koch’s postulates and argued successfully against Alfred Fischer to convince European scientists that bacteria could be plant pathogens. A major bacterial genus, *Erwinia*, was named to honor him; his first name was used because his last name was so common.

Xanthomonas bacteria are yellow, as their name implies (xantho = yellow in Greek), but most plant-pathogenic bacteria are creamy white in culture. Some *Pseudomonas* species fluoresce under ultraviolet light when grown on a specific differential medium, and *Xanthomonas* species produce large amounts of EPS, so the colonies generally look quite fluid and sticky. *Erwinia* species are facultative anaerobes, so they can be identified by sealing them off from a supply of oxygen with wax or oil and observing their growth to distinguish them from aerobic bacteria. Through a series of physiological tests on various media, most plant-pathogenic bacteria can be identified to genus fairly readily.

DISEASE CLASSIC

Soft Rot

Hosts any fleshy fruit, vegetable, or ornamental

Pathogen *Pectobacterium carotovorum* (syn. *Erwinia carotovora*)

Symptoms and Signs Surfaces of fleshy tissues become discolored and depressed, cracked, and begin to exude watery liquid; leaves quickly become dark and slimy.

Disease Cycle This bacterium survives in soil, on contaminated equipment, and on insects that make wounds during feeding. Infection usually occurs in the field, but soft rot also is a common postharvest disease. Additional infections occur from wounding and spreading of bacteria on grading lines, in storage containers, and during improperly performed hydrocooling. Pectolytic enzymes produced by the bacteria degrade the middle lamellae of cell walls and cause tissues to collapse. The bacteria move intercellularly and can quickly destroy large amounts of tissue when conditions are moist and warm.

Management Practice sanitation of tools and storage areas. Avoid wounds of fleshy fruits and vegetables during production and harvest. Manage insects that cause wounds. Avoid dripping water in storage, and keep temperatures cool.

Significance This destructive disease reduces both the quality and availability of fresh fruits and vegetables and our ability to store them for long periods.

Plant Disease Profiles: Blackleg of potato (related disease)

Although many bacteria grow quickly, identification by isolation and growth on a series of media can take too long when a grower has a serious disease problem. It also may be necessary to identify a bacterium to species or to pathovar, rather than to genus only. Rapid identification using immunoassays such as enzyme-linked immunosorbent assay (ELISA) and/or DNA-based PCR tests can be used in these situations (see Appendix 3). Additional tests for bacterial identification include substrate utilization or antibiotic susceptibility tests (e.g., those manufactured by Biolog and Vitek, respectively) and fatty acid methyl esterase (FAME) profiling. These techniques also are important in situations where several different bacteria can cause similar symptoms in a plant.

Accurate diagnosis of bacterial diseases usually requires isolation of the causal agent in pure culture. The genus often can be determined by the Gram stain reaction, number and location of flagella, and growth on selective media or in the absence of oxygen. However, immunoassays and PCR may be the simplest and quickest methods to identify a bacterial species or pathovar.

How are bacterial diseases managed?

Exclusion

Seeds harvested from infected plants are often infested or infected with bacteria. The production of seed in dry climates with few bacterial diseases results in pathogen-free seed that can be planted in humid areas. In the United States, packets of flower and vegetable seeds may be marked as “western-grown” to indicate the decreased risk of disease. Vegetatively propagated plants also may be produced in pathogen-free conditions via tissue culture (see Chapter 11). This reduces the chance of distributing vascular wilt bacteria and fastidious bacteria along with the propagated plant materials. This is extremely important for many greenhouse flower crops, such as chrysanthemums and geraniums, where millions of plants are produced in a short period of time.

Quarantines have been used effectively to delay the spread of some bacterial diseases. For example, a fire blight quarantine was established in Australia in 1924 after the disease was found in New Zealand. Although fire blight has been reported in and then eradicated from Australia once, this quarantine has been quite effective for many decades.

Eradication

Some plant-pathogenic bacteria can be eradicated from an infected host or from the environment before they infect a host. In annual crops, the inability of many plant-pathogenic bacteria to produce spores and their limited survival in soil or debris for long periods is to the grower's advantage. Bacterial populations can be eradicated by **crop rotation** to nonhost plants and can be reduced even more quickly by destruction of infested plant debris and weed hosts. For example, routine weed management can help eliminate sources of the aster yellows phytoplasma from areas around flower and vegetable crops, and removal of nearby wild cherry trees with X disease (also caused by a phytoplasma) can help keep peach and cherry orchards free of this disease. Some plant-pathogenic bacteria are very difficult to eradicate. For example, soft rot bacteria (e.g., *Pectobacterium carotovorum*) are soil inhabitants that persist in the soil and can continue to infect host plants many years after the previous susceptible crop was removed.

Some bacteria can be eradicated from infected host tissue or from the environment by physical and chemical treatments. Heat treatments are the most common method for physical eradication. For example, the fire blight bacterium, *Erwinia amylovora,* cannot survive several hours at temperatures of 45–50°C (113–122°F) and has been eliminated from apple and pear shoots subjected to heat treatments. Some seedborne bacteria can also be eliminated from seed stocks that are treated with hot air or water, but it is important not to harm the fragile plant embryo in the process.

DISEASE CLASSIC

Fire Blight

Hosts apple, pear, and several ornamentals in the rose family

Pathogen *Erwinia amylovora*

Symptoms and Signs Symptoms of fire blight include blossom water-soaking and necrosis, leaf blight, twig necrosis and shepherd's crooks, branch and trunk cankers, and fruit lesions. Numerous diseased shoots make a tree appear burned, hence the name "fire blight." Ooze is a common sign.

Disease Cycle *Erwinia amylovora* overwinters in cankers and is spread to blossoms when insects are attracted to ooze from cankers in the spring. The bacteria survive as epiphytes and eventually invade the flowers through nectaries or the shoots and fruits through wounds created by insects or storms. During the summer, the bacteria spread intercellularly in the tissue, reaching branches and trunks, where cankers develop.

Management Fire blight can be managed by planting resistant cultivars, by roguing trees with cankers, by applying antibiotics (to lessen floral infection), and by pruning blighted shoots 20–25 centimeters (8–10 inches) below the visible end of the canker. Disinfest pruning tools between cuts. Pruning tools should be disinfested between cuts.

Significance Fire blight is a limiting factor in commercial fruit production. For example, fire blight limits pear production in the United States to semiarid areas west of the Rocky Mountains. In addition, *Erwinia amylovora* was first bacterium proven to cause a disease of plants.

Plant Disease Profiles: Fire blight

In perennial woody plants, pruning and destroying twigs and branches with bacterial cankers is a labor-intensive, but effective, method to eradicate bacterial populations. This cultural method is used to manage fire blight in apple and pear orchards. Winter pruning is safer because bacteria are less active in cold, dry weather and so are less likely to be spread by pruning at that time. In routine landscape management, infested tools can spread fire blight bacteria from a single infected branch to many susceptible plants in the rose family such as mountain ash, crabapple, and flowering pears and quince. Pruning tools must be disinfested with alcohol or bleach between cuts. Pruning cuts should be made well beyond the affected area because bacteria may be colonizing tissue below the visible canker. At times, entire orchards must be eradicated to eliminate a disease. Millions of orange trees were cut and burned in the southeastern United States in efforts to manage citrus canker, caused by *Xanthomonas axonopodis* pv. *citri* (Figure 3.17).

► DID YOU KNOW?

Strains of the bacterium that causes citrus canker (*Xanthomonas axonopodis* pv. *citri*) were accidentally introduced into the southeastern United States at least three different times during the 1900s. Each time, millions of citrus trees in nurseries, orchards, and homeowners' yards were cut and burned to eradicate the disease. From 1994 to 2006, more than $1 billion were spent on eradication by federal and state governments. A series of hurricanes in 2004 and 2005 spread the bacterial pathogen across the state, and in 2006 the Florida House of Representatives voted to halt the eradication program.

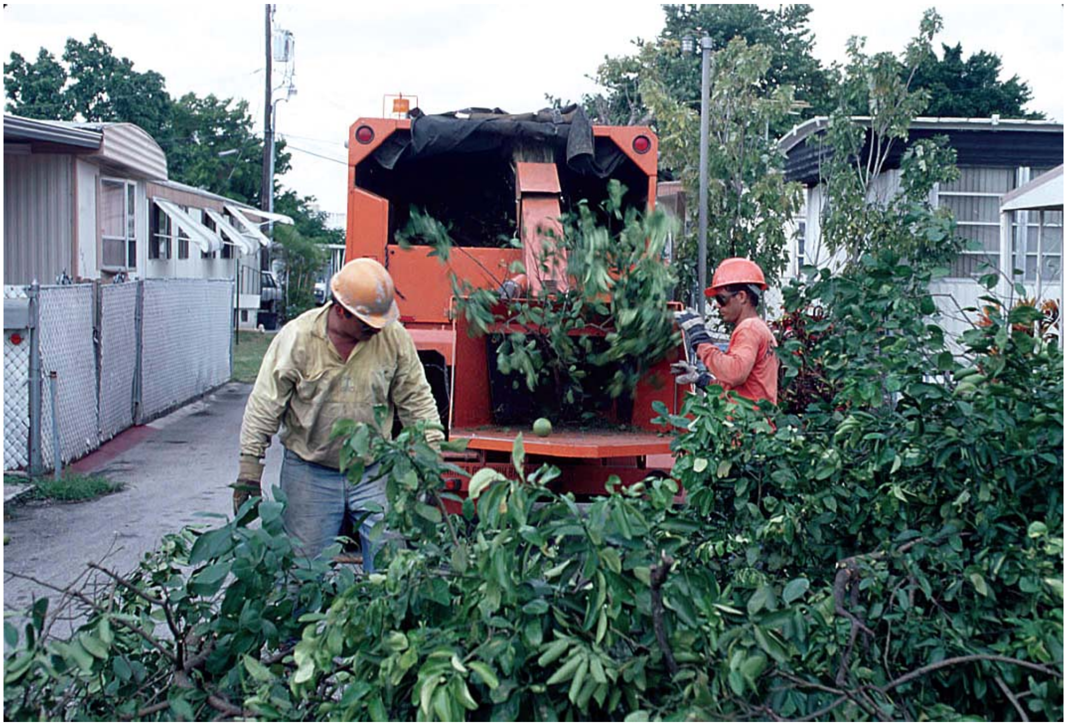

Figure 3.17. Commercial chipping machine used to chip citrus trees infected by or exposed to citrus canker in urban Miami, Florida.

Antibiotics are chemicals that are important for control of many human and animal diseases caused by bacteria, but they play a much smaller role in management of bacterial plant diseases. This is because they tend to be quite expensive, and repeated use, as occurs in humans and animals, results in bacterial populations that are resistant to the antibiotic. Populations of fastidious prokaryotes in some high value trees have been reduced by injection of tetracycline antibiotics, leading to remission of symptoms. These antibiotics are effective against phytoplasma and spiroplasma diseases because they kill bacteria that have no cell walls, unlike penicillin-type antibiotics, which work by preventing bacterial cell wall formation.

Finally, some bacterial diseases can be managed by eradication of their insect vectors, especially if the bacteria overwinter in association with the vectors. For example, overwintering corn flea beetles transmit the bacteria that cause Stewart's wilt of corn (*Erwinia stewartii*, syn. *Pantoea stewartii*), and many fastidious bacteria are transmitted by leafhoppers. Carefully timed applications of insecticides to kill these vectors help to reduce the occurrence of these diseases.

► **DID YOU KNOW?**

In 2006, a citrus disease known as citrus huanglongbing (HLB), or citrus greening, was discovered in Florida. HLB is caused by bacteria in the genus *Candidatus* Liberobacter, which are transmitted by small insects called psyllids. Infected citrus trees have reduced yields and fruit quality and die over several years. There are no curative measures at this time, so citrus-growing areas without HLB have quarantines in place against both the bacteria and the psyllid vectors.

Protection

Chemicals are used much less frequently to protect plants from bacterial infections than they are to manage fungal diseases. The most effective bactericides contain copper (e.g., Bordeaux mixture) and have been used to manage some leaf spots and blights caused by bacteria. However, if the environment is conducive to multiplication and spread of the bacteria, chemicals are unlikely to be effective. Copper-based chemicals, which remain on the plant surface, may help prevent infections, but they cannot be used to cure plants once infection has occurred. Antibiotics also have been used as protectants (Figure 3.18). For example, streptomycin has been sprayed in apple and pear orchards to prevent infection by fire blight bacteria. Concerns about the development of resistant pathogen populations have reduced the use of this management strategy.

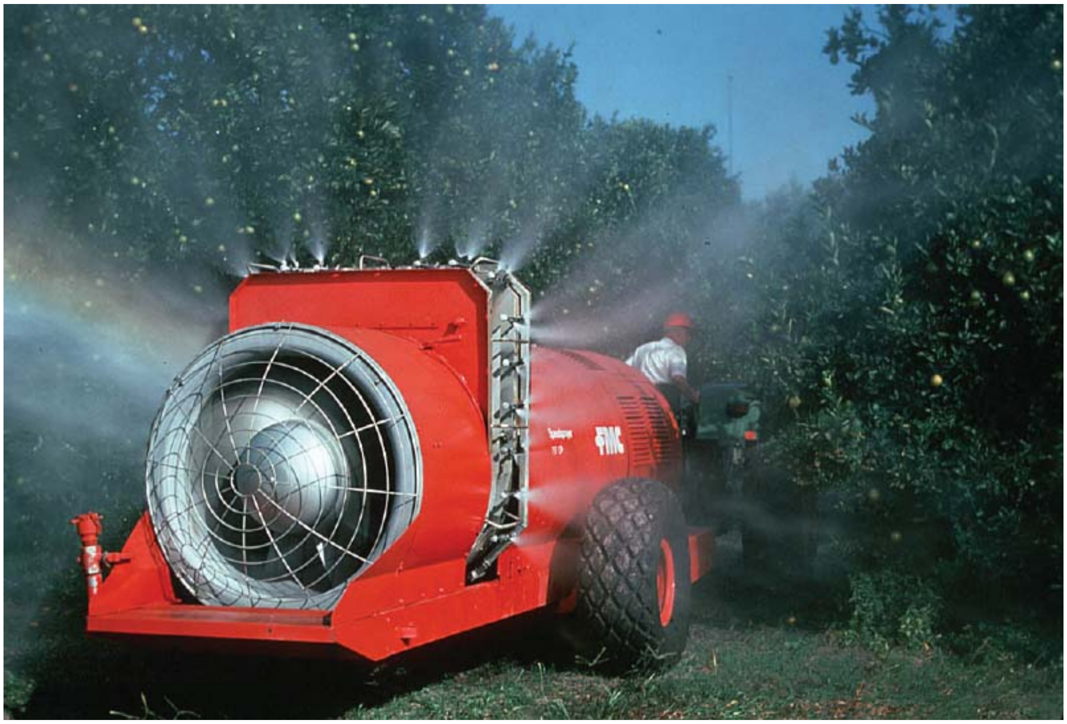

Figure 3.18. Typical air-blast sprayer for application of copper-based sprays or antibiotics.

► DID YOU KNOW?

The Centers for Disease Control and Prevention (CDC) estimate that 50 million pounds of antibiotics are produced annually in the United States. About half of the antibiotics are used by humans in prescriptions from doctors, and 40% are used for animal diseases and production. The combined use of streptomycin and oxytetracycline on plants accounts for about only 0.1% of all antibiotics produced annually in the United States. Nearly all of this is applied to fruit trees (apples and pears) for management of fire blight (*Erwinia amylovora*).

A number of cultural practices can help protect plants from infection. For example, foliar bacterial pathogens can be spread rapidly by splashing water and the movement of people and machinery through plantings, especially when plants are wet. Overhead irrigation should be applied when plants can dry quickly. Agricultural activities such as cultivation should be delayed, when possible, until plants are no longer wet. Surface irrigation, in which water is distributed over the soil surface, helps minimize leaf wetness. Other methods that speed plant drying and reduce bacterial multiplication include removing weeds, using wider spacing between individual plants and between rows, and orienting rows with respect to predominant air movement. Windbreaks and other barriers can reduce windblown rain that carries bacteria from plant to plant. Foliar bacterial diseases are more severe in the soft, lush tissues of over-fertilized plants, so excessive applications of nitrogen fertilizers increase plant susceptibility.

► DID YOU KNOW?

Bacteria in potato tubers infected with ring rot disease (*Clavibacter michiganensis* subsp. *sepedonicus*) can contaminate the surfaces of mechanically cut seed potatoes and cause serious losses. There is a zero tolerance for this disease in seed potatoes due to its highly destructive nature.

► **DID YOU KNOW?**

In May, 2000, fire blight killed 350,000–450,000 apple trees in southwestern Michigan. Apple yields were reduced by 35% over the region, and some growers suffered 100% losses in some plantings. Recovery of yields was estimated to take at least 5 years, with cumulative losses of nearly $36 million. Besides the optimal environmental conditions that year, this epidemic was caused by high-density plantings, highly susceptible cultivars, the presence of bacterial strains resistant to streptomycin, and a late spring freeze.

Biological management methods have been developed for some diseases caused by plant-pathogenic bacteria. Crown gall is one example. *Agrobacterium tumefaciens* has a very large host range but is particularly important on stone fruits and woody ornamentals. Roots of these plants can be treated with selected strains of the related nonpathogen *Agrobacterium radiobacter* during transplanting. The protecting strains compete with *A. tumefaciens* for space on the surface of the plant roots and produce an antibiotic that is specifically toxic to *A. tumefaciens*, thus killing the bacteria and reducing infection. Considerable research is in progress to find other antagonistic bacteria that will protect plants from infection by pathogenic bacteria (see Chapter 11).

DISEASE CLASSIC

Crown Gall

Hosts members of 93 families of plants, including pome fruits, stone fruits, nuts, ornamentals, and vines and canes

Pathogen *Agrobacterium tumefaciens*

Symptoms and Signs Plants with crown gall develop tumorous overgrowths called galls. Crown galls typically form on the root "crown" of the plant near the soil line but also can form on roots or on aboveground stems and twigs. The galls are initially small and usually white or tan, more or less round, and spongy in texture. As the galls enlarge, the outer tissue gradually darkens to brown and becomes convoluted and rough, and the inner tissue becomes hard. On perennial hosts, the galls are perennial and often grow to about 2.5 centimeters (1 inch) in diameter.

Disease Cycle *A. tumefaciens* can survive in soil as a saprophyte for short periods (but survives for much longer in the rhizosphere of many crop and weed hosts) before it invades a host plant through a wound. Once within the plant, the bacterium induces formation of a gall by transferring a piece of its plasmid DNA into the cell of the plant, where it becomes integrated into the plant's genome. Genes on this tumor-inducing (Ti) plasmid cause the plant cell to divide repeatedly, thus forming the tumor, and to produce compounds that are used by the bacterium as food. The bacteria live and multiply in the intercellular spaces of the gall and are released back into the soil when the gall eventually deteriorates.

Management Crown gall can be managed by avoiding infested fields, by planting certified disease-free plants or crown gall-resistant plants, by treating roots of young transplants with antagonistic strains of *Agrobacterium radiobacter*, and by roguing infected plants. Tools used for grafting or pruning should be disinfested frequently.

Significance Crown gall is an economically important disease on a wide range of hosts. Also, the Ti plasmid of *A. tumefaciens* is commonly used to introduce desirable genes into plants (genetic engineering). (See Chapter 9.)

Plant Disease Profiles: Crown gall

Host plant resistance is a very important management strategy for bacterial diseases, but resistant cultivars must be selected for each different disease (Figure 3.19). Resistance may be due to physical barriers, such as a thicker cuticle or greater numbers of trichomes, or to chemical barriers, such as inhibitors. If the resistance is due to only one gene in the host plant (single gene resistance), new strains of bacteria may quickly evolve that can overcome the resistance and infect the previously resistant host.

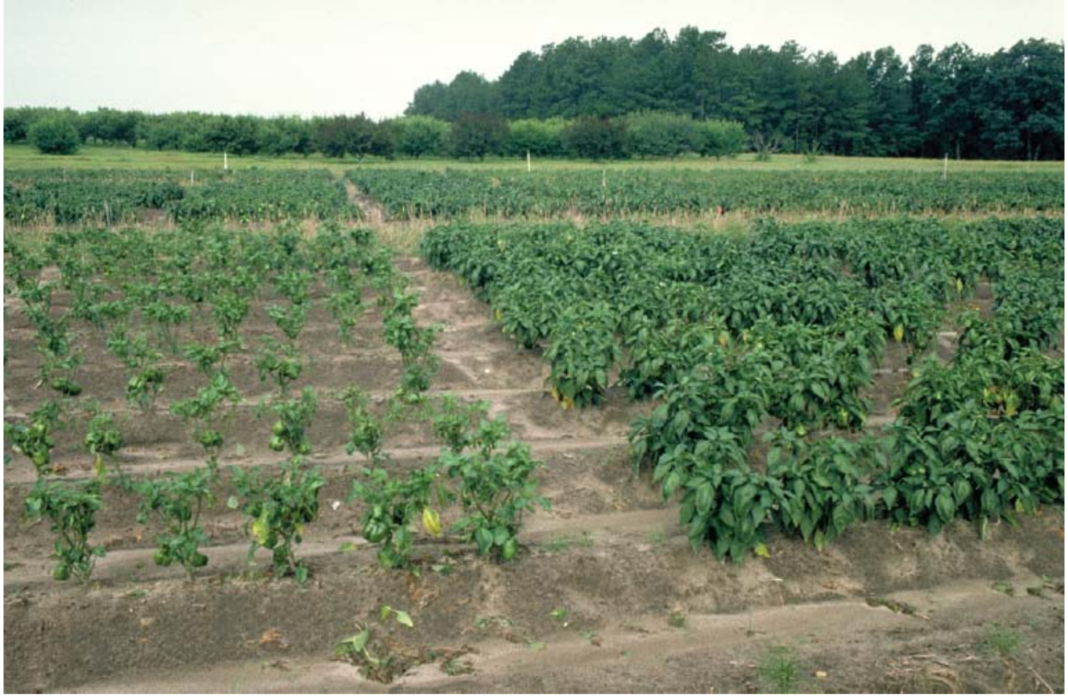

Figure 3.19. Pepper plants susceptible (left) and resistant (right) to specific strains of the leaf spot bacterium, *Xanthomonas axonopodis* pv. *vesicatoria*.

The complete genome sequences of some plant-pathogenic bacteria have been determined, and such studies are under way for a number of others. This information may lead to the identification of novel resistance genes that can be incorporated into plants through genetic engineering.

► **DID YOU KNOW?**

A rice gene for resistance to rice blight, caused by *Xanthomonas oryzae* pv. *oryzae*, has been genetically engineered into rice cultivars that are grown on over 9 million hectares (22 million acres) of rice in Asia and Africa.

Molecular biologists are beginning to understand the details of host-pathogen interactions in bacterial diseases. It has long been known that the ability of bacteria to multiply quickly under favorable conditions is a key to their success as plant pathogens. Modern research suggests that bacterial populations actually communicate with each other (**quorum sensing**) during infection and colonization. This knowledge may lead to new approaches to management (see Chapter 11).

Management of bacterial diseases of plants is often difficult and complex, requiring a combination of strategies. Host plant resistance and crop rotation are widely used management strategies, but eradication of bacteria in host plants or in vector populations and exclusion of bacteria from planting seed or stock also are important.

Management Strategies for Bacterial Diseases

Exclusion

- Impose quarantines (local and international).

- Grow plants in dry environments.
- Plant pathogen-free seed or stock.

Eradication

- Rotate to nonhosts.
- Rogue weed hosts.
- Destroy infested plant debris.
- Use heat treatment of seeds or propagative material.
- Apply antibiotics.
- Apply insecticides (to kill infective vectors).

Protection

- Apply copper-based chemicals.
- Apply antibiotics.
- Apply bacterial antagonists.
- Minimize leaf wetness.
- Plant resistant hosts.

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Questions

1. Compare the sizes, shapes, and genome compositions of a typical bacterium and fungus that infect plants. How is genetic variation introduced into the populations of these two types of plant pathogens?
2. What are some important differences between common bacteria and fastidious bacteria as plant pathogens?
3. What advantages do many fungi have as plant pathogens compared with bacteria? Consider various stages of the disease cycle, including survival, dispersal, and infection.
4. What symptoms and signs suggest infection by a bacterial pathogen? What are some typical symptoms that you would expect to see in a plant infected by (a) a xylem-limited fastidious bacterium and (b) a phytoplasma or a spiroplasma?
5. Would you expect to see bacterial streaming if you cut (a) a twig from an apple tree with fire blight or (b) a gall from a rose with crown gall? Explain why or why not.
6. Bacterial diseases are more common in warm, wet climates. What are some ways that bacteria can survive in winter conditions or dry seasons?
7. What are the principal means of spread of plant-pathogenic bacteria from one host to another? How are these similar to or

different from the principal means of spread of plant-pathogenic fungi?

8. Of the bacterial diseases described in this chapter, which are monocyclic and which are polycyclic in a typical growing season?
9. Explain and give an example of a strategy used to manage bacterial diseases in each of the following categories: eradication, exclusion, and protection.
10. Explain why insecticides are sometimes used to protect high value crops from infection by phytoplasmas but are not used in apple orchards for fire blight.

Challenge questions

1. *Erwinia amylovora* (fire blight) was accidentally introduced into England in 1957 and into Europe in 1966 and has spread from there. Using your knowledge of the environmental conditions of these areas, what do you expect the impact of fire blight to be on apple and pear production in these areas?
2. Soft rot bacteria can destroy almost any fleshy plant product in storage. They are found in nearly every soil. What practices at harvest and in the preparation and maintenance of a cold storage facility will improve the shelf life of potatoes, squash, or lettuce?
3. You have been shipped a set of culture plates containing bacterial isolates for use in a resistance screening program, but the labels have come off. You know that the set includes isolates of *Xanthomonas, Erwinia, Pseudomonas*, and *Clavibacter*. What laboratory tests can you do to identify these isolates to genus?
4. Alfred Fischer and Erwin Smith engaged in a classic and bitter debate in the late nineteenth century regarding the existence of

bacterial diseases of plants. Given the times and what you know about bacterial diseases, why was this so controversial?

Supplementary Resources

Illustrated Glossary

Special Topics

- Bacteria as plant pathogens
    - Las bacterias como patógenos vegetales (Spanish)
- Fastidious vascular-colonizing bacteria
    - Bacterias fastidiosas colonizadoras vasculares (Spanish)

Plant Disease Profiles

- Bacterial fruit blotch of cucurbits
- Bacterial leaf scorch
- Bacterial spot of pepper and tomato
- Blackleg of potato
- Citrus canker
- Crown gall
- Fire blight of apple and pear
- Lethal yellowing of palms
- Stewart’s wilt of corn

Laboratory Exercise

Screening for active ingredients in plant extracts that inhibit the growth of *Agrobacterium tumefaciens*

► **WORDS TO KNOW**

The words in this list are defined in the Illustrated Glossary.

- actinomycete
- aerobic
- anaerobic
- antibiotic
- bacilliform
- bacterial streaming
- bacteriophage
- bacterium (pl. bacteria)
- capsule
- cell wall
- chemotaxis
- coccus
- colony
- conjugation
- crop rotation
- dilution plating
- dilution streaking
- epiphyte
- eradication
- exclusion
- extracellular polysaccharide (EPS)
- fastidious
- fission
- flagellum (pl. flagella)
- gall
- genome
- gram-negative
- gram-positive
- Gram stain

hydathode
intercellular
intracellular
latent period
lenticel
middle lamella (pl. middle lamellae)
mollicute
mutation
nectary
nucleoid
ooze
pathovar (pv.)
peritrichous
phloem necrosis
phytoplasma
plasmid
pleomorphic
polar
prokaryote
propagative transmission
protection
quorum sensing
ribosome
saprophyte
scorch
selective medium
soft rot
spiroplasma
stoma (pl. stomata)
stylet
subspecies
toxin
water-soaked

wilt
witches' broom
wound
xylem-limited fastidious bacteria
yellows

Exercises

Exercise 3.1. Label the structures on the drawing of a bacterial cell.

Exercise 3.2. Identify the symptoms and signs of bacterial diseases.

Internet Resources

APS Resources

Agrobacterium: The natural genetic engineer, 100 years later
Antibiotic use for plant disease management in the United States
Bacterial blight of anthuriums: Hawaii's experience with a global disease
Bacterial scorch of shade trees
Citrus huanglongbing: The pathogen and its impact
Citrus huanglongbing: Understanding the vector-pathogen interactions for disease management
Phytoplasma casts a magic spell that turns the fair poinsettia into a Christmas showpiece
Post-hurricane analysis of citrus canker II: Predictive model estimation of disease spread and area potentially impacted by various eradication protocols following catastrophic weather events
Ralstonia solanacearum race 3 biovar 2 causes tropical losses and temperate anxieties

Recommended Website

Bacterial Nomenclature Up-to-Date

Internet Research

Internet Research Exercise 3.1. Using the APS *Common Names of Plant Diseases*, select a bacterial disease of an annual herbaceous plant. Consult the Internet and other resources to find the answers to the following questions:

a. What is the name of the bacterium?
b. How does the bacterium survive adverse environmental conditions?
c. What host plants does the bacterium infect?
d. How is the bacterium disseminated horizontally (plant to plant) and vertically (to the next generation)?
e. What management practices are recommended?

Internet Research Exercise 3.2. Find a fact sheet or disease profile of a bacterial disease on a plant of interest. What are the symptoms and signs of the disease? How is diagnosis confirmed (i.e., how is the bacterium identified?) What diseases caused by other pathogens might be confused with this disease?

CHAPTER 4

What Are the Causes of Plant Diseases?

► Nematodes

IN THIS CHAPTER:

- ► **What are nematodes?**
- ► **What symptoms and signs do nematodes cause?**
- ► **How do nematodes survive and spread?**
- ► **How are nematode diseases diagnosed?**
- ► **How are nematode diseases managed?**

What are nematodes?

Nematodes are nonsegmented roundworms. They are the most abundant multicellular animals on earth and are found in every soil, every body of water, and even in the extreme environments of deserts and the polar ice fields. Their ubiquitous presence was eloquently described by Nathan Cobb, the father of U.S. nematology (see quote). Over three billion nematodes can be present in an acre of land. Because of their abundance, they are an important component of every ecosystem.

> “In short, if all the matter in the universe except the nematodes were swept away, our world would still be dimly recognizable, and if, as disembodied spirits, we could then investigate it, we should find its mountains, hills, vales, rivers, lakes, and oceans represented by a film of nematodes. The location of towns would be decipherable, since for every massing of human beings there would be a corresponding massing of certain nematodes. Trees would still stand in ghostly rows representing our streets and highways. The location of the various plants and animals would still be decipherable, and, had we sufficient knowledge, in many cases even their species could be determined by an examination of their erstwhile nematode parasites.”
>
> —From “Nematodes and Their Relationships,” 1915, Nathan Cobb, “father of U.S. nematology”

Most nematodes feed on bacteria, other small life forms, and organic matter. Many of these nematodes (about 25% of the known species) are soil-dwelling; more (about 50% of the known species) are found in marine sediments. Some specialized nematodes (about 15%) are animal parasites causing human diseases such as elephantiasis, pinworm, and trichinosis. The intestinal worms of pets and heartworm in dogs are nematodes. Other specialized nematodes (about 10%) are plant parasites (Figure 4.1). All of the plant-pathogenic nematodes are biotrophs (obligate parasites), requiring a living host plant for survival.

► DID YOU KNOW?

The largest nematode identified (*Placentonema gigantissima*) lives in the placenta of the sperm whale and can reach up to 8 meters (26 feet) in length!

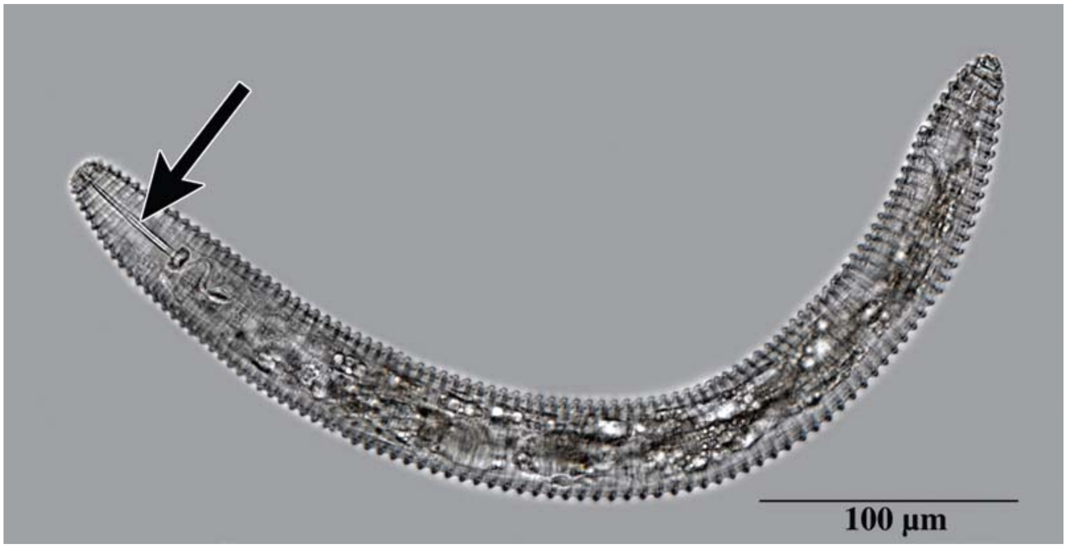


Figure 4.1. A ring nematode (*Mesocriconema xenoplax*). Note the stylet, the feeding structure (arrow).

Some of the larger animal parasites were described many centuries ago, and the history of plant-pathogenic nematodes began quite early too. There are reports of a soybean problem in China in 235 B.C. that may have been caused by the soybean cyst nematode (*Heterodera glycines*). Turbeville Needham, a clergyman, recorded his observations of seed gall nematodes (*Anguina tritici*) in a wheat grain in 1743. He crushed the infected grain in water and used a microscope to observe the wiggling nematodes.

► DID YOU KNOW?

Some estimates of nematode damage to crops worldwide approach $100 billion a year, and this does not include the cost to manage the nematodes.

Despite these early observations, there was little scientific progress in the study of nematodes as plant pathogens until 1855. Another clergyman, M. J. Berkeley, who first proposed that a fungus was the cause of late blight of potatoes, also discovered root-knot nematodes (*Meloidogyne* spp.) (Figure 4.2). Many previous nematode observations were reinterpreted as the germ theory became accepted, and people began to recognize nematodes, along with fungi and bacteria, as causes of plant disease.

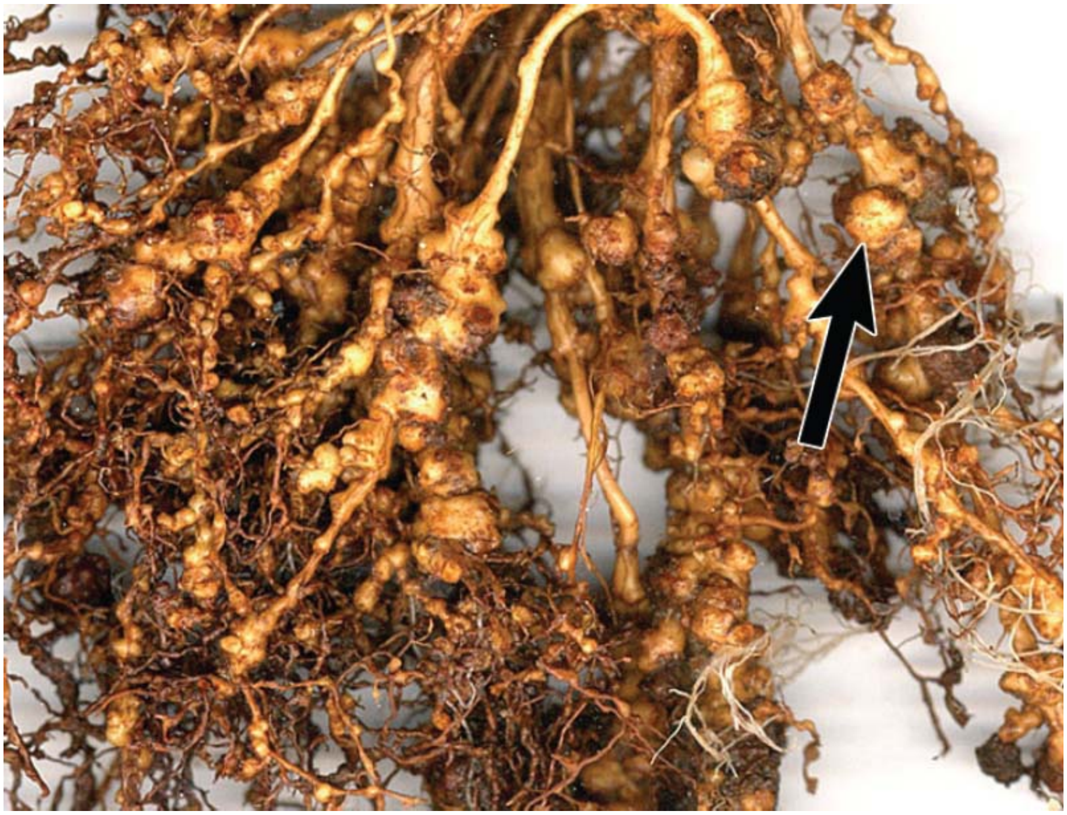


Figure 4.2. Closeup of root knots (galls) (arrow) on lettuce caused by root-knot nematodes (*Meloidogyne* spp.).

Plant-pathogenic nematodes are very small; most range from 300 to 1,000 micrometers in length. A few are barely visible to the unaided eye in water or on microscope slides. Thus, it is not possible to observe them without magnification in soil or in plants.

Nematodes feed on plants with a **stylet**, a specialized feeding structure that resembles a sharp, hollow needle. Muscles are attached to the stylet. The nematode uses its stylet to penetrate plant tissues, probe during feeding, secrete polysaccharides and proteins, and ingest the cellular contents of plant cells.

Nematodes have a simple life cycle. They develop into complete worms within **eggs** before they hatch and grow through four **juvenile** stages to become reproductive **adults** (Figure 4.3). (Some references refer to juveniles as *larvae*. However, larvae, e.g., caterpillars, undergo metamorphosis. Juveniles look like adults but are not able to reproduce.) Like insects, nematodes shed their outside **cuticle** and part of the lining of the stylet during the process of **molting**. The first molt of all plant-parasitic nematodes occurs in the egg. Although the second-stage juvenile is most often the stage that infects the plant, other stages, including adults, may serve as the infective stage for many species. The final stage is the sexually mature adult that may be male or female (Figure 4.4). A single female of some species can lay hundreds of eggs. The time from egg to mature adult can be as short as a few weeks but is affected by environmental conditions.

Plant-pathogenic nematodes are nonsegmented roundworms that begin as eggs and progress through four juvenile stages until they become adults capable of reproduction. The second-stage juvenile is the infective stage for most nematodes that are plant pathogens. All plant-pathogenic nematodes feed with a stylet. They are biotrophs (obligate parasites) of plants. Some species are ectoparasites; others are endoparasites. Within these two groups are both migratory and sedentary species. A few nematode species are vectors of plant viruses; all of these are migratory ectoparasites.

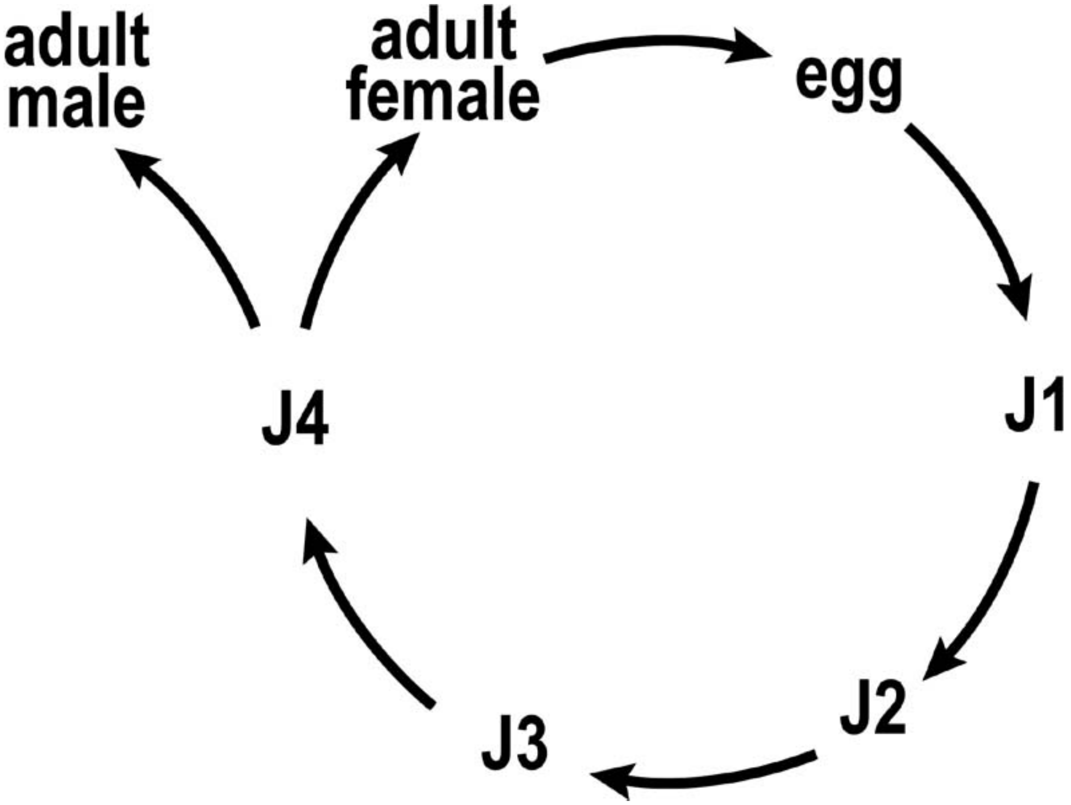
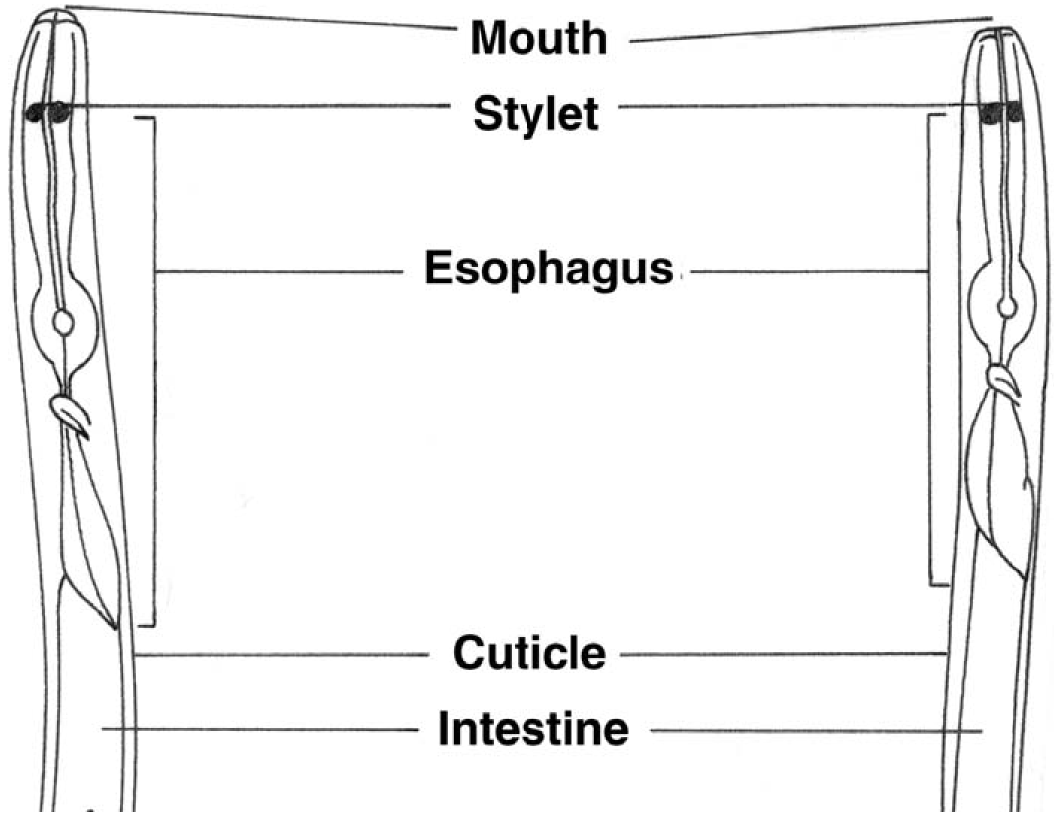

adult male
adult female
egg
J1
J2
J3
J4

Figure 4.3. Life cycle of a nematode including four juvenile (J1–J4) stages.


Mouth
Stylet
Esophagus
Cuticle
Intestine

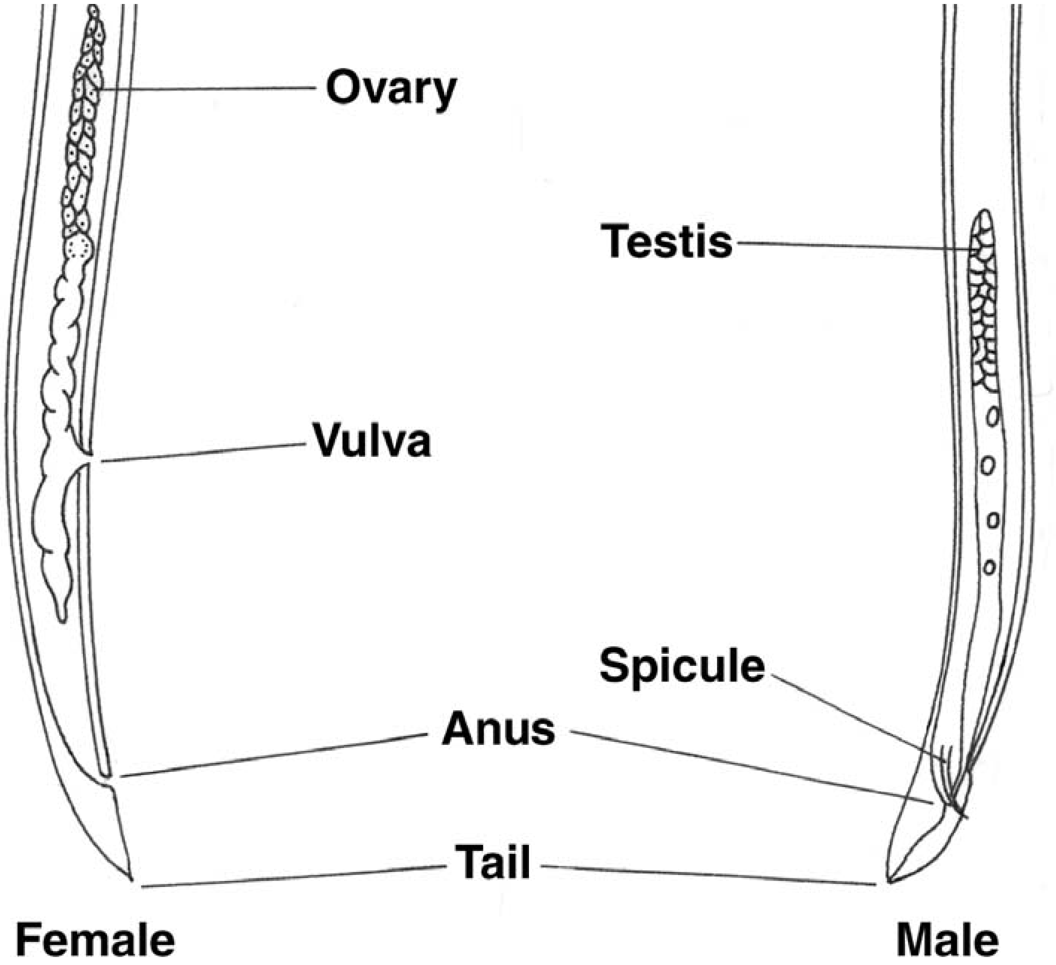


Figure 4.4. Generalized nematode anatomy.

In some nematode populations, environmental conditions such as food availability or temperature affect sex ratios. Females may dominate a population, but males become more common under stressful conditions. In some species of plant-pathogenic nematodes, reproduction is **parthenogenetic** (not requiring males for fertilization of eggs).

Plant-pathogenic nematodes feed on plant tissues in different ways. Some feed with their probing stylets from the outside of plant tissue as **ectoparasites**. Most of the ectoparasites are **migratory**, moving from cell to cell as they feed, but some remain **sedentary** and feed from specialized plant cells in feeding sites induced by the nematode. Many ectoparasitic species cause little obvious injury to plant roots, but a few cause severe damage to root tips.

Migratory ectoparasites may move from plant to plant through the soil. Some are **vectors** of plant viruses, which cause damage that may overshadow the damage done by the nematodes as they feed on the roots. The stylets are important in virus transmission, as they are for aphids and related insect virus vectors. The nematodes acquire and transmit certain viruses when feeding.

In contrast, other species of nematodes are **endoparasites**, which remain on the same plant and enter plant tissue to feed from the inside. **Migratory endoparasites** continue to feed while moving through plant tissue, killing cells as they go and leaving dead and dying cells behind. **Sedentary endoparasites** induce the formation of specialized feeding sites and remain there until they die. Some additional types of nematodes do not fit exactly into these simplified categories.

► DID YOU KNOW?

Almost half the genes identified in the human genome (43%) match genes identified in the bacteria-feeding nematode *Caenorhabditis elegans*. This was the first animal to have its complete genome sequenced. It now serves as a model species for biological research.

Types of Plant-Pathogenic Nematodes

Ectoparasites feed from outside the plant

- **Migratory**: moves, feeding, from plant to plant
- **Sedentary**: remains to feed on same plant, perhaps using specialized feeding structures

Endoparasites feed from inside plant tissues

- **Migratory**: moves within and feeds on tissues of same plant

- **Sedentary**: remains within same plant tissue and feeds at specialized feeding sites

► **DID YOU KNOW?**

In Australia, livestock that graze in fields of annual ryegrass with seedheads can suffer from a neurological toxicity disorder. *Anguina* nematodes may carry the bacterium *Clavibacter toxicus* to the seedheads of the grass plants. The bacteria produce a toxin in the nematode-infected seed galls only when the bacteria are infected by a bacteriophage (bacterial virus). By unknown mechanisms, the virus enhances the ability of the bacterium to produce the toxin. Hay contaminated with infected seedheads also is toxic to animals.

► **DID YOU KNOW?**

Three different Nobel prizes have been awarded to scientists who study the bacteria-feeding nematode *Caenorhabditis elegans* so far! The awards were for groundbreaking discoveries in animal development, genomics, and programmed cell death (2002), **RNA interference (RNAi) for gene silencing** (2006) (see Chapter 9), and the use of the jellyfish green fluorescent protein (GFP) gene as a biological marker (2008).

► **DID YOU KNOW?**

Some nematodes (e.g., *Heterorhabditis* and *Steinernema*) are pathogens of insects and are considered “beneficial nematodes." They can be purchased and used as biological controls for soil insect larvae (rootworms) that damage the roots of crops, ornamentals, and turfgrasses.

What symptoms and signs do nematodes cause?

Nematodes are aquatic, so the plant pathogens among them are primarily pathogens of belowground plant parts, especially roots. Because most nematodes are soilborne pathogens, the most common aboveground symptoms are those of a plant with an impaired root system: **wilting, stunting, yellowing**, and other symptoms that resemble symptoms of nutrient deficiency. Nematodes tend to be found in "hot spots" in fields, somewhat circular areas of poorly growing plants where nematode populations are high (Figure 4.5). Circular patches of diseased plants occur with many soilborne pathogens and are not specific to nematodes. A more specific diagnosis requires examination of the soil, roots, and other underground plant parts to determine the cause.

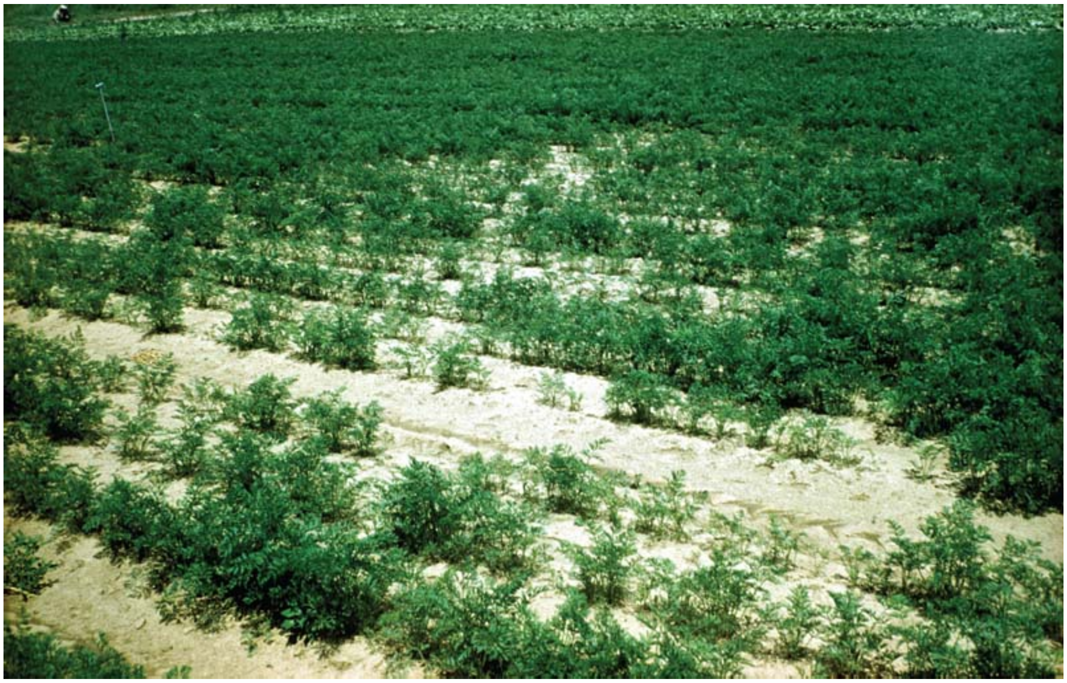


Figure 4.5. Typical symptoms of nematode damage in the field include stunted plants in a somewhat circular pattern (or "hot spot"). (These are carrots infected by lesion nematodes.)

Some nematode common names (**foliar, lesion, root-knot**) describe the symptoms they cause or where the plant becomes infected. Some ectoparasites have names that describe the stylet (**dagger**) or other nematode anatomy (such as coarse ridges, or **rings**, on the cuticle). The following short descriptions illustrate some common types of plant-pathogenic nematodes (Figure 4.6).

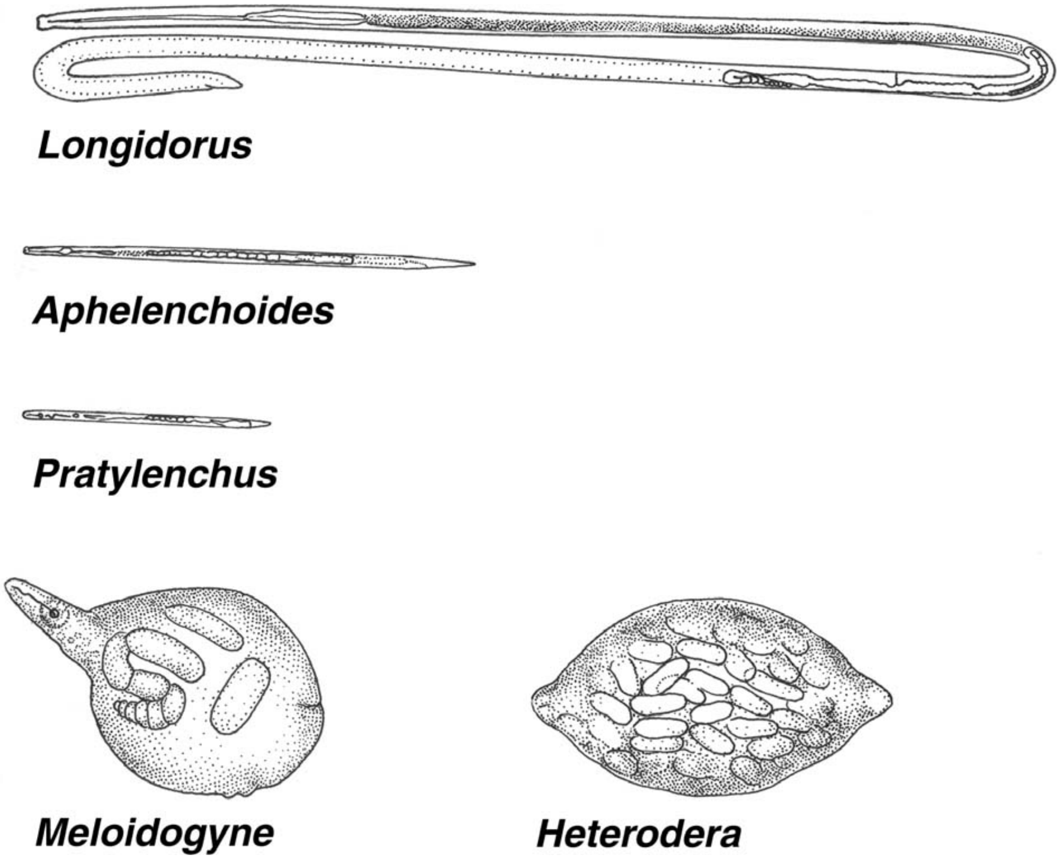

Longidorus
Aphelenchoides
Pratylenchus
Meloidogyne
Heterodera

Figure 4.6. Morphology and relative size of some important plant-pathogenic nematodes.

► DID YOU KNOW?

The nematode "guinea worms" (*Dracunculus*), like those unleashed from polar ice in the 1990s movie "Smilla's Sense of Snow," were first described in the ancient Egyptian scrolls, "Papyrus Ebers," in 1550 B.C.

Nematodes That Infect Plants Below Ground

ROOT-KNOT NEMATODES

Root-knot nematodes (*Meloidogyne* spp.) are named for the galls or "knots" they induce after infection of host plant roots (Figure 4.2). These nematodes are **sedentary endoparasites**. Second-stage juveniles penetrate just behind the root cap and induce permanent feeding sites called **giant cells** within the galls (Figure 4.7). Giant cells form in response to nematode secretions. The plant cell nucleus continues to divide, but cell division does not occur, and the cells begin to swell. Some feeding juveniles grow and molt to become round adult females that lose their **vermiform** (worm-shaped) shape. Females lay a mass of eggs in a gelatinous substance on the root gall surface (Figure 4.8). This substance helps the eggs resist desiccation.

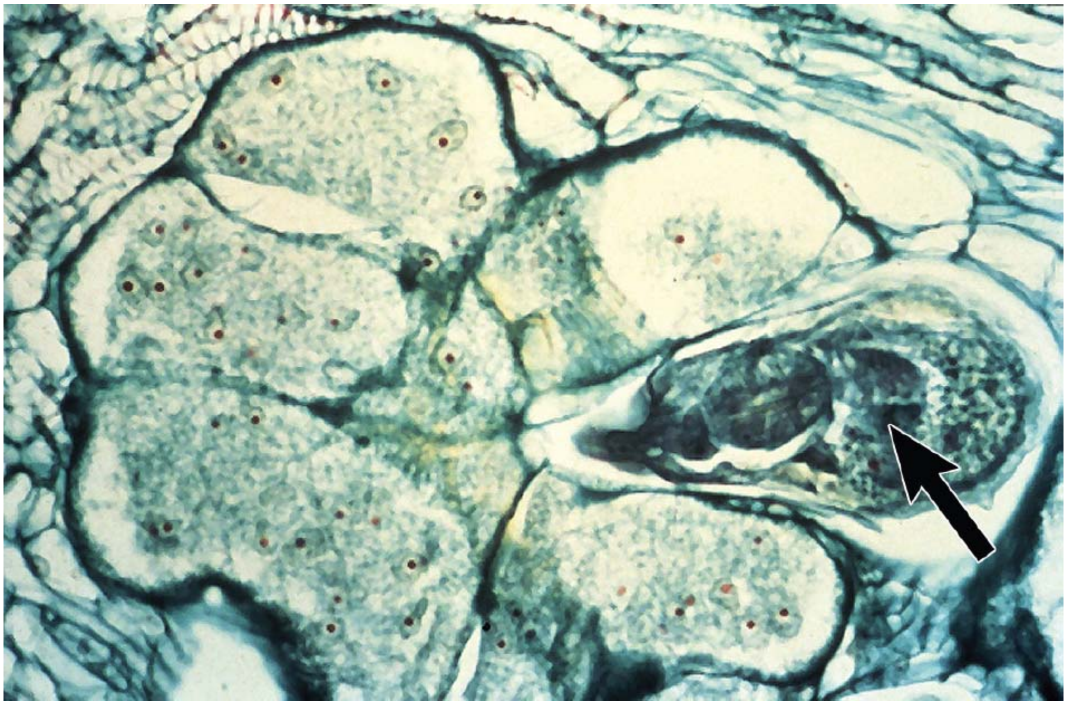


Figure 4.7. Cross section of the giant cell system initiated by a female root-knot nematode (arrow).

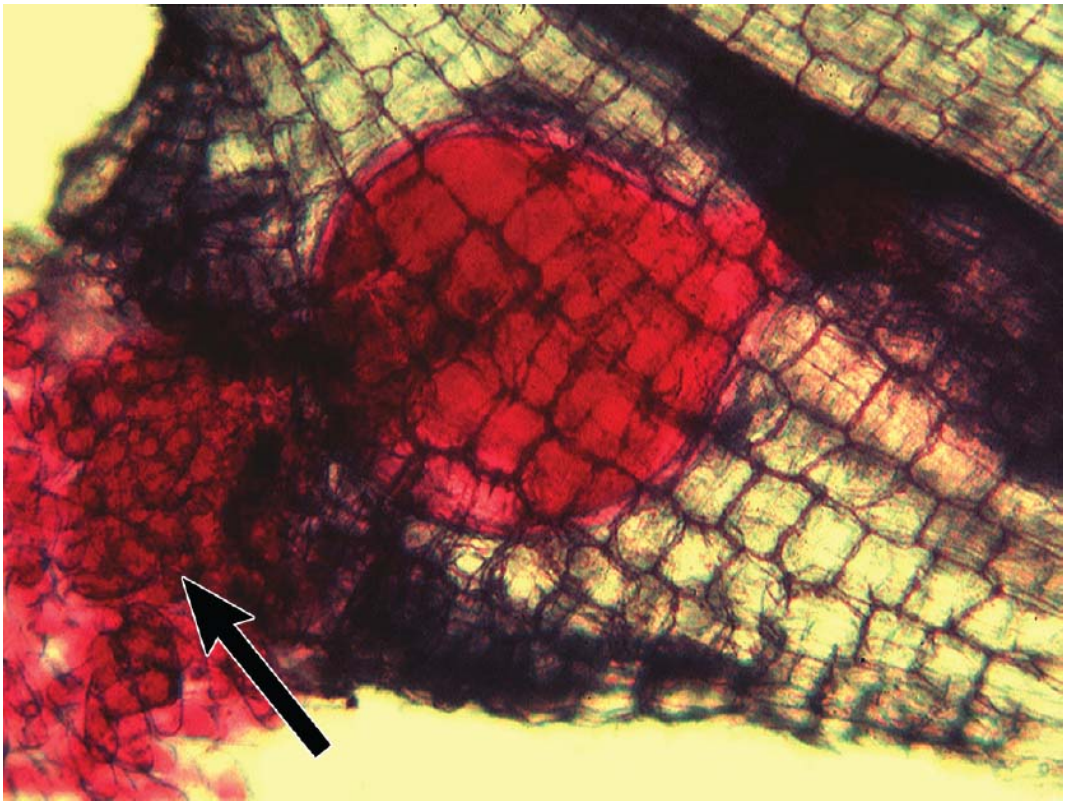


Figure 4.8. Root-knot nematode female with egg mass (arrow).

Root-knot nematodes have **wide host ranges** (i.e., they are not host specific). Root-knot nematode disease is one of the most familiar nematode problems because the root symptoms are easily visible and because the nematodes are widespread and difficult to manage. Worldwide, root-knot nematodes cause more crop losses than any other genus of nematode. Root-knot nematodes are important pathogens of many field, flower, and vegetable crops.

► DID YOU KNOW?

A single plant cell transformed into a "giant cell" for feeding by a root-knot nematode can contain 100 nuclei.

DISEASE CLASSIC

Root-Knot Nematode Disease

Hosts hundreds of species, including fruit crops, grasses, vegetable crops, and numerous weeds

Pathogen *Meloidogyne* spp. (endoparasitic, sedentary)

Symptoms and Signs Above ground, plants (often in a somewhat circular patch) are stunted, may wilt easily, and show nutrient deficiency symptoms. Below ground, numerous small galls or "knots" are visible on roots.

Disease Cycle Second-stage juveniles infect plant roots. Secretions from the nematode's stylet induce the formation of a permanent feeding site with giant cells and the development of a gall. Root-knot nematodes are endoparasitic and become sedentary. Adult females become swollen and may break through the root epidermis. Eggs are laid in a gelatinous mass (see Figure 4.8).

Management Resistant cultivars are available for a number of food and fiber crops. Crop rotation may be used if an acceptable nonsusceptible crop is available. Chemical treatments are sometimes used.

Significance Root-knot nematodes are the most destructive nematodes worldwide on a wide range of plants.

Plant Disease Profiles: Root-knot nematodes

CYST NEMATODES

Cyst nematodes (*Heterodera* spp.; *Globodera* spp.) are similar to root-knot nematodes in that the adult female is swollen rather than vermiform, and they are **sedentary endoparasites**. Juveniles induce a feeding site that is similar to the root-knot giant cells but is composed of **syncytia**. A syncytium forms from the dissolution of the cell walls between several root cells rather than from the swelling and lack of division of a single cell in giant cells caused by root-knot nematodes. The swelling cyst female eventually protrudes from the plant root. Cysts are the diagnostic sign for this type of nematode. Cyst nematode females retain most of their eggs within their bodies instead of laying a mass of eggs outside. When the female dies, her outer body wall becomes a tough, leathery **cyst** that protects the eggs, sometimes for years (Figure 4.9).

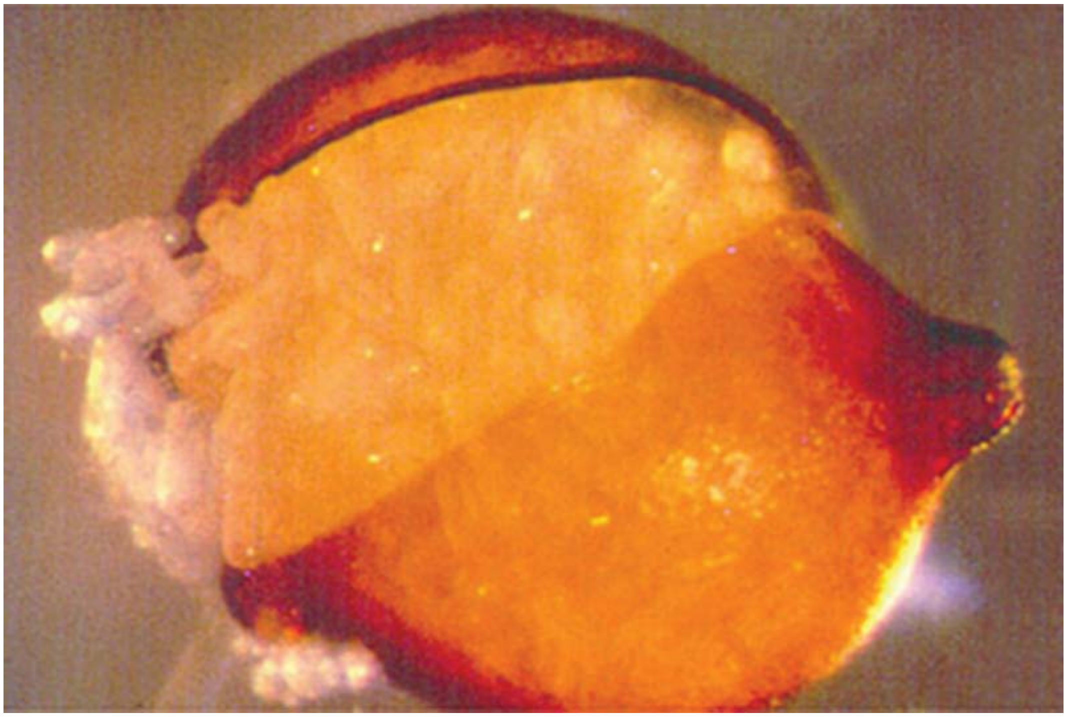


Figure 4.9. Brown cyst of a soybean cyst nematode filled with eggs.

Cyst nematodes are generally **host specific**. Important cyst nematode hosts include cereals, potato, soybean, sugar beet, and tobacco, each of which is infected by different cyst nematode species.

DISEASE CLASSIC

Soybean Cyst Nematode Disease

Hosts soybean (economic host), other legumes

Pathogen: *Heterodera glycines* (endoparasitic, sedentary)

Symptoms and Signs Above ground, stunted plants often are in a somewhat circular patch, may wilt easily, and show symptoms that resemble nutrient deficiency. Yield losses may occur without obvious symptoms. Roots do not usually have any distinctive symptoms, but pinhead-sized, whitish, swollen females and darker mature cysts may be observed on the root surface. Nodulation and secondary roots may be reduced.

Disease Cycle Second-stage juveniles infect plant roots and induce formation of a feeding site with syncytia. Females become swollen and eventually break through the root epidermis. Hundreds of eggs are produced and provide inoculum for secondary infections. Later-produced eggs are retained within the female. Her body becomes a protective cyst that encases viable eggs for years.

Management Resistant soybean cultivars are available. Crop rotation is effective because the soybean cyst nematode is host specific. Management involves a combination of crop rotation and use of resistant cultivars because soybean cyst nematodes can adapt quickly to resistant cultivars. In some areas, egg population densities are monitored, and management is initiated based on economic damage thresholds.

Significance The soybean cyst nematode is presently the most damaging pathogen of soybean in the United States and 10 other countries with major production.

Plant Disease Profiles: Soybean cyst nematode disease

Example of Soybean Cyst Nematode Damage: Estimates for Kentucky

Eggs per 100 cubic centimeters of soil	Potential yield loss
1–500	0.5%
501–1,500	5–15%
1,501–3,000	15–20%
3,001–5,000	20–50%

► DID YOU KNOW?

Soybean cyst nematodes (*Heterodera glycines*) are apparently native to Asia, where the soybean originated. They were first reported from North Carolina in 1954, possibly arriving in soil clinging to flower bulbs imported from Japan. The nematodes spread rapidly to other soybean-growing states. This may have happened because, in the late 1800s (before soybean cyst nematode was recognized as a plant pathogen), soil was imported from Asia to obtain inoculum of the bacterium *Bradyrhizobium japonicum*, which is responsible for nitrogen fixation in soybean. The soil was distributed to researchers working on soybean improvement and to farmers who wanted inoculum of nitrogen-fixing bacteria for their soybean fields.

LESION NEMATODES

Lesion nematodes (*Pratylenchus* spp.) are **migratory endoparasites** (Figure 4.10). They are named for the lesions formed by the dead and dying cells they leave behind as they feed and move through the roots and other underground plant parts. The dying cells are often invaded by fungi and bacteria, which contribute to the destruction of the roots. Many species of lesion nematodes thrive in a wide variety of climates. They have **wide host ranges**.

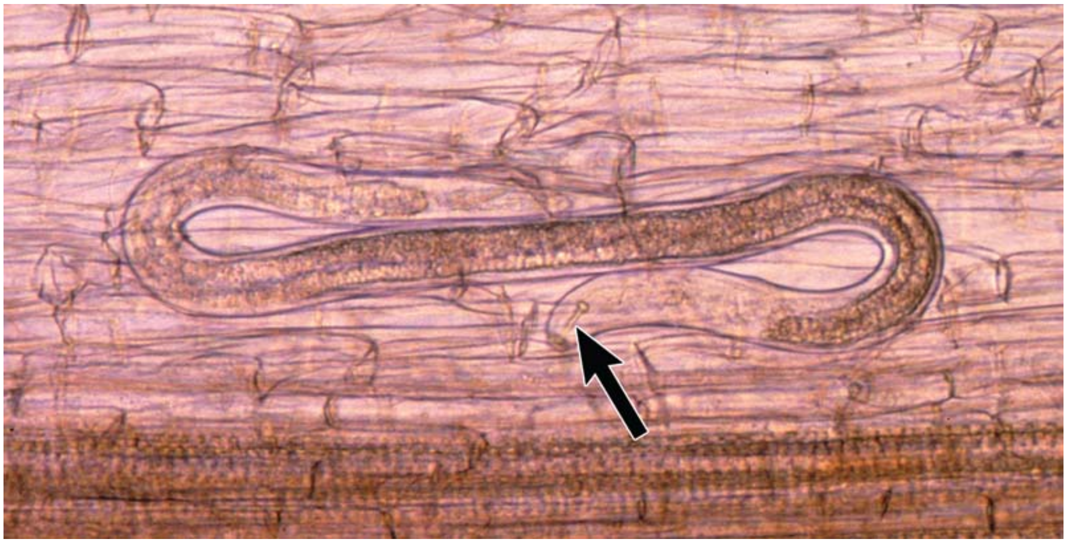


Figure 4.10. Lesion nematode (*Pratylenchus* sp.) in a plant root. Note the stylet, the feeding structure (arrow).

DISEASE CLASSIC

Lesion Nematode Disease

Hosts over 400 host plant species, including fruit crops, monocots, peanut, and potato

Pathogen *Pratylenchus* spp. (endoparasitic, migratory)

Symptoms and Signs Above ground, plants (often in a somewhat circular patch) are stunted, may wilt easily, and show nutrient deficiency symptoms. Infected roots have lesions of dead cells caused by the migratory, endoparasitic nematodes. Other pathogens may invade the lesions and cause disease complexes.

Disease Cycle Adults and all juvenile stages can infect plant roots. They move through internal root tissues, feeding on root cells and killing them. Hundreds of individual eggs are laid by the females both inside and outside the infected roots.

Management Resistant cultivars are not usually available for host plants. Crop rotation may be difficult because of the lesion nematodes' wide host range, including many weeds. Chemical treatments may be used for high value crops.

Significance The broad distribution and wide host range of lesion nematodes make them third in economic importance after root-knot and cyst nematodes.

Plant Disease Profiles: Lesion nematode disease

BURROWING NEMATODES

Burrowing nematodes (*Radopholus* spp.) are similar to lesion nematodes; they are **migratory endoparasites**. Widely distributed in tropical and subtropical climates, they infect a wide range of important crops, including banana, citrus, coffee, and sugarcane.

Common Root-Infecting Endoparasites

sedentary

- cyst (*Heterodera* spp., *Globodera* spp.)
  female becomes cyst of eggs
  syncytia in feeding site
  host specific
- root-knot (*Meloidogyne* spp.)
  galls (knots) on roots
  giant cells in feeding site

wide host range

semi-endoparasitic

- reniform (*Rotylenchulus reniformis*)
  female is kidney-shaped
  syncytia in feeding site
  wide host range

migratory

- burrowing (*Rodopholus* spp.)
- lance (*Hoplolaimus* spp.)
- lesion (*Pratylenchus* spp.)

MIGRATORY ECTOPARASITIC NEMATODES

Migratory ectoparasitic nematodes may have common names such as **dagger** (*Xiphinema* spp.) or **sting** (*Belonolaimus* spp.) that refer to the nematode stylets (Figure 4.11). **Stubby-root**, the common name of *Trichodorus* and its relatives, refers to the symptoms these nematodes induce. Many ectoparasites have **wide host ranges**, and, for some species, all life stages are infective.

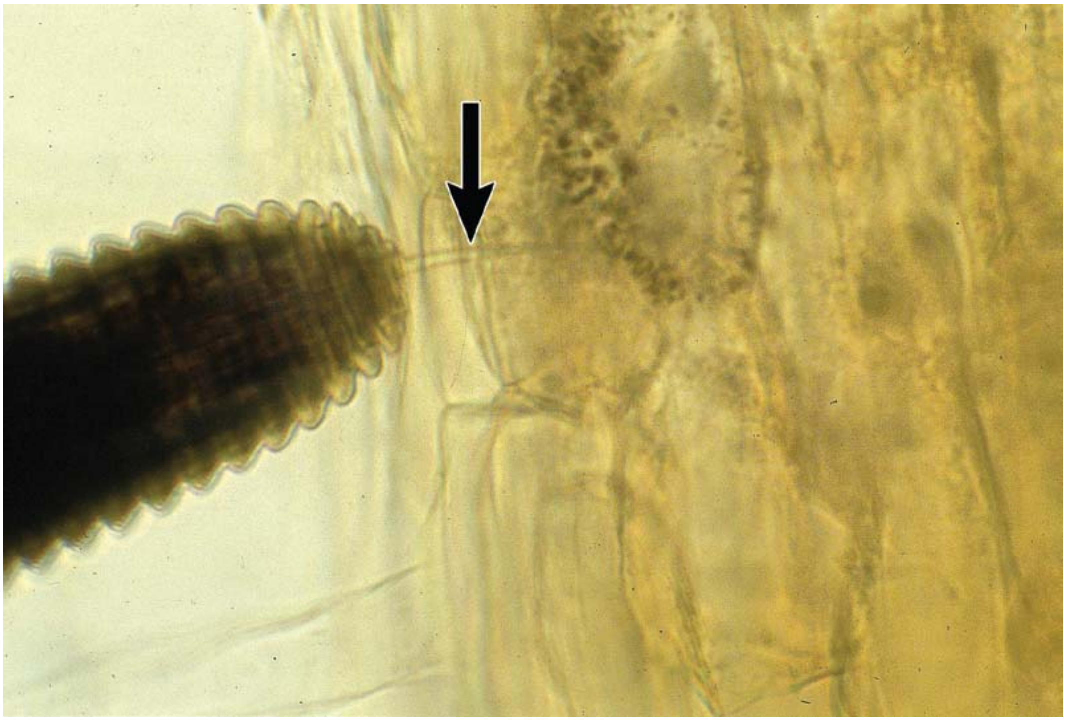


Figure 4.11. An ectoparasitic ring nematode (*Mesocriconema xenoplax*) feeding on a root. Note stylet (arrow).

Diseases caused by ectoparasites may go undiagnosed because they cause nonspecific symptoms of reduced growth. They can be detected only after extraction from soil and examination under a microscope. Some ectoparasites cause significant injury at relatively low numbers, but others can be present in large numbers without doing serious harm. If ectoparasitic nematodes feed in one position long enough, they may cause distortion and reduction in root growth that can be observed (e.g., stubby-root). The impact of ectoparasitic nematodes is often related to other stress factors affecting a plant.

Common Root-Infecting Ectoparasites

- dagger (*Xiphinema* spp.)
- needle (*Longidorus* spp.)
- pin (*Paratylenchus* spp.)
- ring (*Criconemoides* spp.,

Mesocriconema spp.)
- spiral (*Helicotylenchus* spp., *Rotylenchus* spp.)
- sting (*Belonolaimus* spp.)
- stubby-root (*Paratrichodorus* spp., *Trichodorus* spp.)
- stunt (*Tylenchorhynchus* spp.)

Some root-infecting nematodes are classified as **sedentary ectoparasites** or as **semi-endoparasites**, depending on how far they penetrate plant tissue and whether a specialized feeding cell forms. Examples include the **citrus nematode** (*Tylenchulus semipenetrans*), the **reniform** (kidney-shaped) **nematode** (*Rotylenchus reniformis*), and some of the **spiral nematodes** (*Helicotylenchus* spp.).

Nematodes That Infect Plants Above Ground

FOLIAR NEMATODES

Foliar nematodes (*Aphelenchoides* spp.) are **migratory** and move from the soil to the lower leaves of plants in a film of water. As they feed, they often cause distinctive symptoms of fans or wedges of dead cells between leaf veins (Figure 4.12). The injury progresses upward to younger leaves as the nematodes migrate to new tissue. Foliar nematodes may invade leaves through stomata. Most species of foliar nematodes have **wide host ranges**.

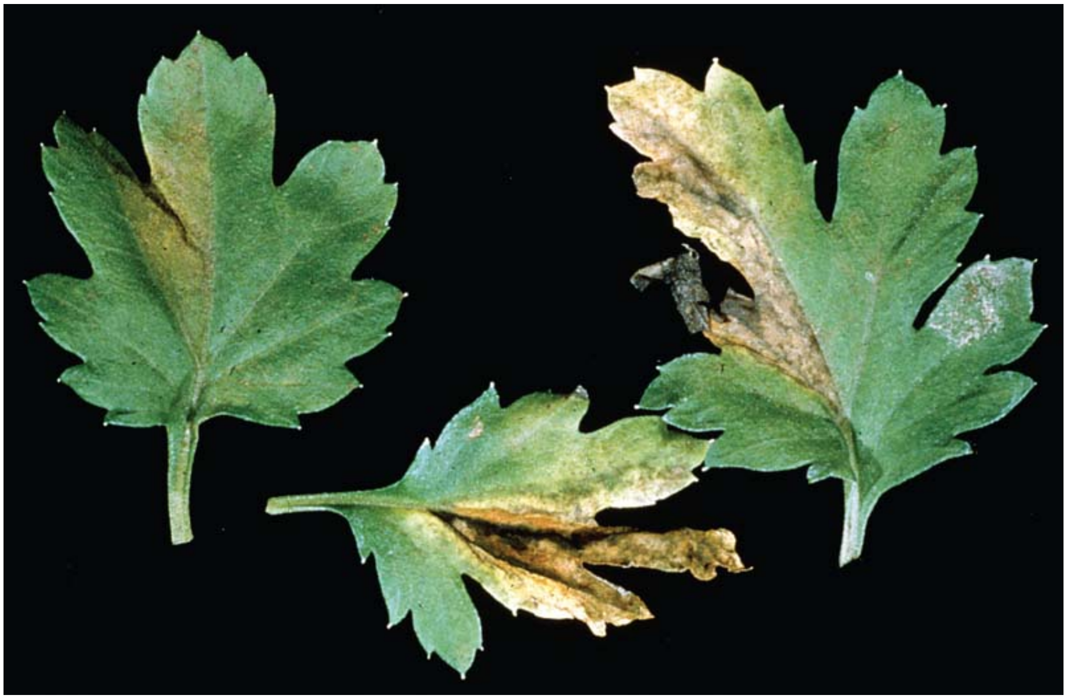


Figure 4.12. Feeding damage on chrysanthemum from foliar nematodes (*Aphelenchoides* spp.). Note leaf lesions bound by major leaf veins.

SEED GALL NEMATODES

Seed gall nematodes (*Anguina* spp.) are **migratory** and cause stem and seed galls on grasses, but a few infect dicots. By entering and feeding on developing seeds, they cause blackened "cockles" (seed galls) filled with nematodes that replace the developing seeds in the seed head.

STEM AND BULB NEMATODES

Stem and bulb nematodes (*Ditylenchus* spp.) are **migratory** nematodes that commonly invade onion bulbs and many flower bulbs through stomata in the bulb scales. The invaded bulb scales may become necrotic. Masses of thousands of dried, viable worms sometimes accumulate as "nema wool" on the bulb surface. Stems and flowers from infected bulbs may be stunted and distorted. Other nematodes in this group can cause deformation and decline of alfalfa, clover, phlox, and strawberries.

Most plant-pathogenic nematodes are root pathogens and cause similar aboveground symptoms of wilting, stunting, and nutrient deficiency. Below ground, root-knot and stubby-root nematodes cause distinctive root symptoms. Ectoparasites may cause distortions in root growth or no root symptoms other than overall decline. Cysts on roots of host plants are signs of cyst nematode infection. Foliar, seed gall, stem and bulb, pine wood, and red ring nematodes feed on aboveground plant parts, causing a variety of symptoms in their hosts.

Common Aboveground Nematodes

- foliar (*Aphelenchoides* spp.)
- pine wood (*Bursaphelenchus xylophilus*)
- red ring (*Bursaphelenchus cocophilus*)
- seed gall (*Anguina* spp.)
- stem and bulb (*Ditylenchus* spp.)

OTHER NEMATODES

A few specialized nematodes are pathogens of trees. **Red ring disease** of coconut palm is caused by a nematode (*Bursaphelenchus cocophilus*). In this disease, nematodes remain relatively confined to a red band of stem tissue, usually evident in cross section about a meter (several feet) above the soil line. The palm weevil and other insects are vectors of this nematode. Another example is the **pine wood nematode** (*Bursaphelenchus xylophilus*), which has caused severe **pine wilt** epidemics in Japan. Cerambycid or sawyer beetles (*Monochamus* spp.) are vectors of the pine wood nematode. This nematode is thought to have originated in North America because pines native to North America are relatively resistant to it. Pine wilt can kill pine trees native to Asia or Europe in a single season.

► **DID YOU KNOW?**

Genes that encode enzymes that degrade plant cell walls (cellulases) appear to have been acquired from soil bacteria and incorporated into the genome of today's plant-pathogenic nematodes.

► **DID YOU KNOW?**

Turbatrix aceti is a nematode that lives in vinegar. This "vinegar eelworm" used to be an unexpected guest at some dinner tables and is still sometimes seen today.

How do nematodes survive and spread?

Most nematodes lack specialized survival structures. Thus, populations may decline quickly in the absence of a host plant or in soils dried by drought or winter freezing. Unfortunately, perennial plants, including both crops and weeds, can continue to support nematodes even in winter. Survival in association with various perennial weeds is especially important for the many nematodes with wide host ranges. People contribute to the survival of nematodes by storing infected propagative plant parts (such as bulbs, corms, tubers, and rootstocks) and then introducing the nematodes into the soil along with the planting material.

Nematodes can survive as eggs in the soil even though they are vulnerable during the winter and in dry soils. Their populations typically decline significantly over the winter months. The eggs of cyst nematodes are protected from desiccation within the leathery cuticle of the dead female body. The tough cysts offer some additional means of dispersal, including in windblown, infested soil and long-distance travel in the digestive systems of migratory birds. Eggs of some cyst species require a hatching stimulant from host root exudates. This requirement prevents premature hatching of these host-specific nematodes when no host is available. The eggs of most nematode species do not require such a stimulant to hatch.

Stem and bulb nematodes may accumulate in visible masses called "nema wool." The outer nematodes die and form a coating that reduces desiccation of the inner nematodes. During unfavorable environmental conditions (cold, drought, or heat), these nematodes and many others are capable of **cryptobiosis**, a reversible state in which the nematode's metabolism nearly stops. Some seed gall nematodes were found to be still viable after 25 years in a cryptobiotic state. Despite these specialized adaptations, survival remains a challenge for these tiny pathogens.

Nematodes may survive on perennial plants or propagative parts of plants, as eggs or cysts, or in a cryptobiotic state. Many nematodes die during droughts or in winter. Nematodes may be dispersed by water, propagative parts, machinery, and other means of soil movement. Some tree-infecting nematodes have insect vectors.

Nematode Survival Mechanisms

- cryptobiosis
- cysts (*Heterodera, Globodera*)
- eggs in soil
- perennial plants, including weeds
- propagative plant parts

Nematodes can move only short distances, using snakelike motions, in the thin water layer that coats soil particles. Sandy soils, having larger particles, allow some nematodes to move greater distances. However, long distance movement of nematodes occurs mostly by human activities, such as the movement of propagative parts (bulbs, rootstocks, tubers), soil on equipment, and irrigation water.

Rain and overhead irrigation aid in the movement of foliar, stem and bulb, and seed gall nematodes from soil and plant debris to new host leaves, stems, and flowers. Splashing water can disperse the nematodes between plants over short distances.

Nematode Dispersal Mechanisms

- active movement through films of water
- insect vectors (some tree pathogens)
- machinery, boots
- propagative parts
- water

A few plant-pathogenic nematodes are tree pathogens that are transmitted by insects. Pine wood nematodes are carried in the tracheae of the sawyer beetle's (Figure 4.13) respiratory system and emerge when the beetle feeds on a new host. The nematodes then colonize the new host, causing pine wilt. They also may travel long distances in wood chips or unseasoned lumber. Similarly, several weevil species are vectors of the red ring nematode, leading to red ring disease of coconut palms.

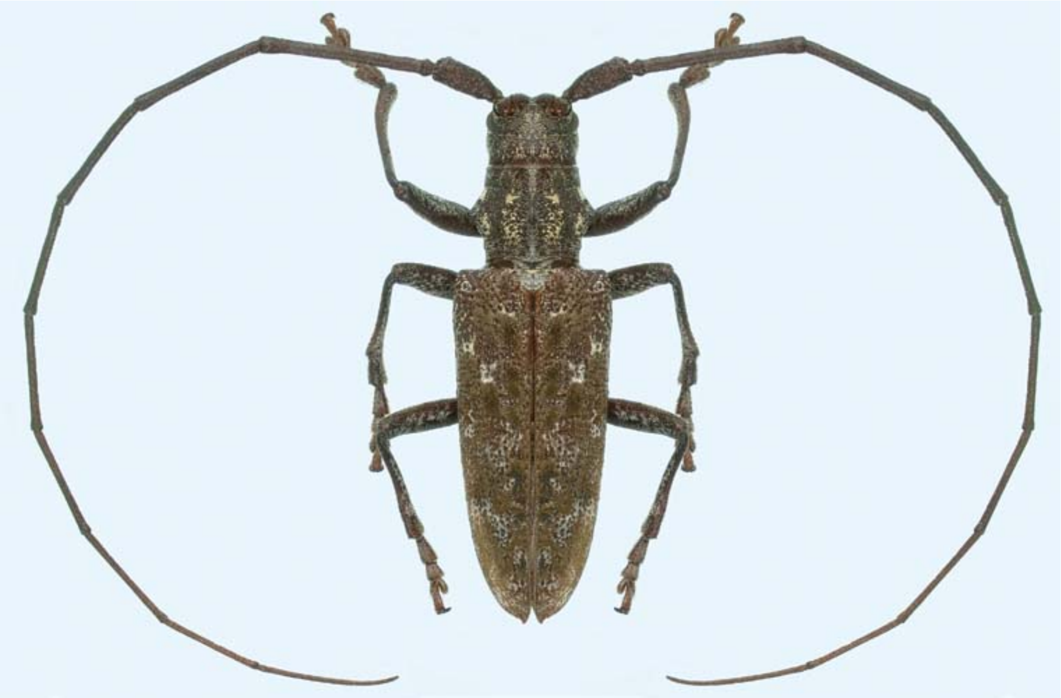


Figure 4.13. Pine sawyer beetle (*Monochamus carolinensis*), vector of the pine wood nematode (*Bursaphelenchus xylophilus*), which causes pine wilt disease.

How are nematode diseases diagnosed?

The most common aboveground symptom of a nematode problem is an overall decline of plants in a localized area. Nematodes can cause significant decline in crop yields, however, even without visible aboveground plant symptoms. It is common for several kinds of plant-pathogenic nematodes (including both ectoparasites and endoparasites) to be active on a plant at the same time. Diseases caused by nematodes are often difficult to diagnose because the symptoms are so similar to those caused by certain soilborne insects, nutrient and water deficiencies, vascular wilts, and fungal and bacterial root pathogens (Figure 4.14). In fact, the damage caused by root-feeding nematodes is often seen together with that caused by other root-infecting organisms, which may complicate the diagnostic process.

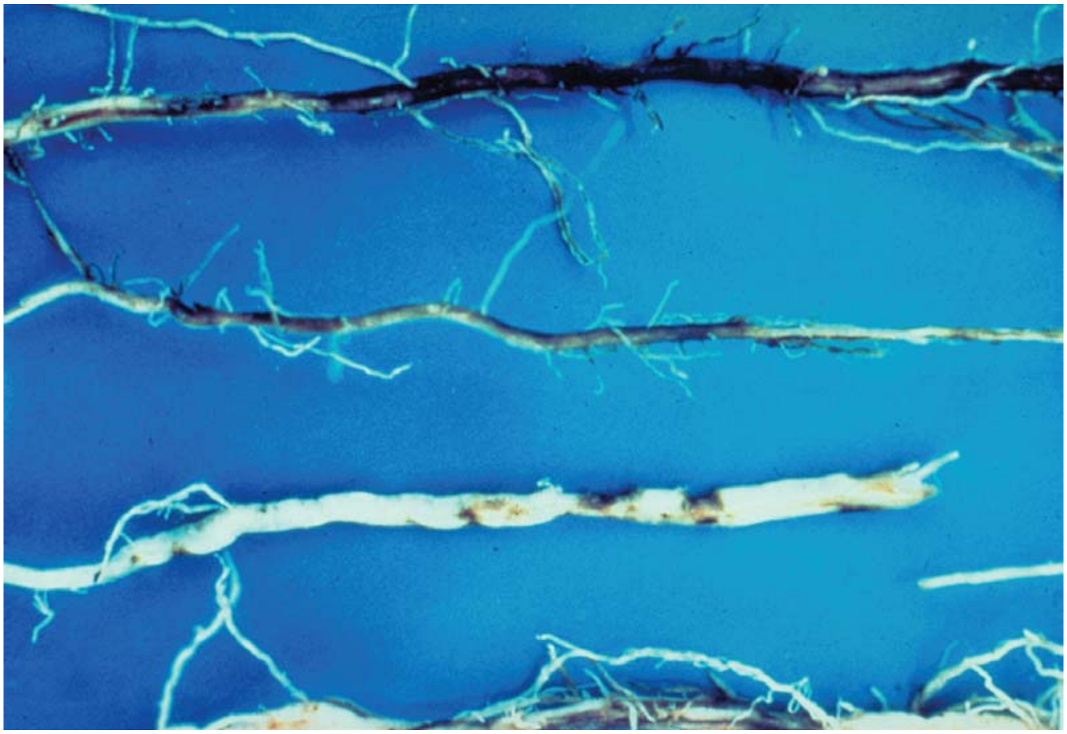


Figure 4.14. Necrotic lesions on corn roots infected by lesion nematodes (*Pratylenchus* spp.).

It may be possible to diagnose a few nematode-induced plant diseases by careful examination of infected plants. Galls induced by root-knot nematodes, cyst nematode females on the root surface (Figure 4.15), stubby roots, “nema wool” (for stem and bulb nematodes), and areas of interveinal leaf necrosis filled with nematodes (for foliar nematodes) are some of the relatively few specific signs and symptoms of nematode damage to plants.

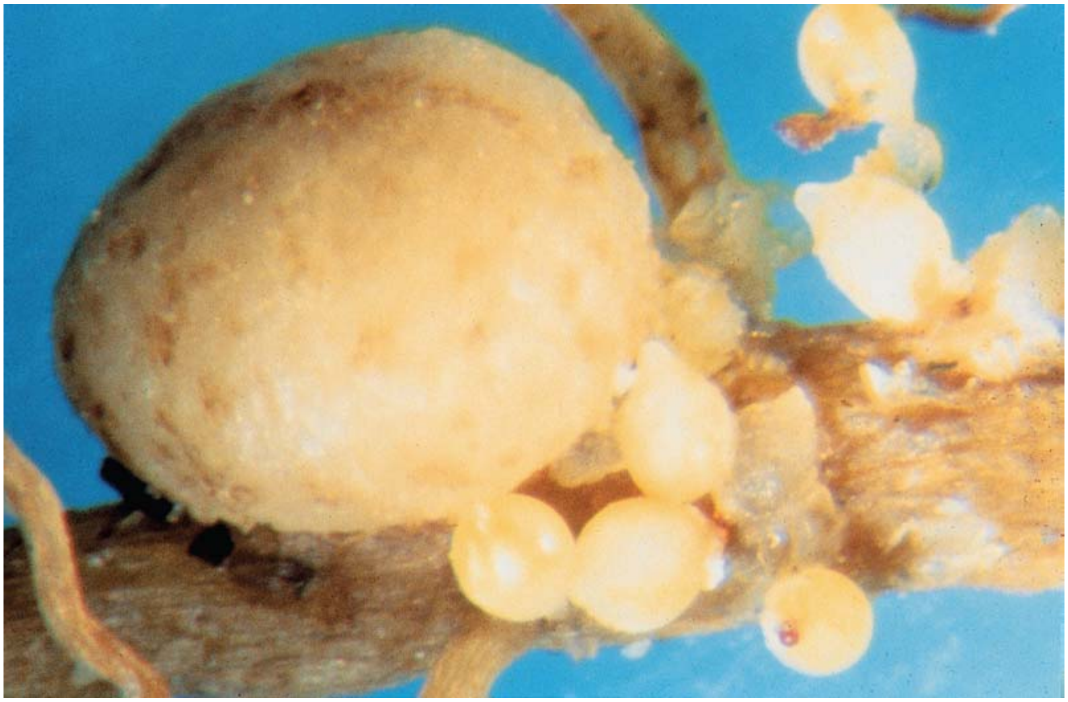


Figure 4.15. Soybean cyst nematode females (which appear as small, whitish-yellow bodies) erupting through the soybean root surface. The large nodule contains nitrogen-fixing bacteria.

Nematodes that infect aboveground plant parts (foliar, pine wood, red ring, seed gall, and stem and bulb nematodes) can be diagnosed by teasing apart some affected tissues while looking through a dissecting microscope. It is common to see numerous nematodes present in these tissues, but only those with stylets are potential plant pathogens.

Extracts from soil and plant material must be examined under a microscope to determine whether root-infecting plant-pathogenic nematodes are present. To properly diagnose nematode problems, however, samples of both soil and plant material of affected plants are generally recommended. Nematodes are extracted from measured soil samples into water in several ways, but then they must be identified under a microscope, usually to genus, and counted to determine the number per volume or weight of soil or plant sample. They must be counted because, as with insects, it is their numbers that determine the severity of the problem. This usually requires a laboratory with an experienced nematologist.

Plant-pathogenic nematodes are identified by their general size and shape, the appearance of the stylet (slender, stout, short, or long), the tail (round or pointed), the surface of the cuticle (smooth or ridged), and other morphological features. Most plant-pathogenic nematodes can be identified to genus with a microscope. Most nematode diagnostic keys are based upon characteristics of the adult life stages, but many of these traits are present in the juvenile life stages that make up the majority of individuals extracted (especially from soil).

If the pathogenicity of a nematode must be proven, a modification of Koch’s postulates is used. As biotrophs (obligate parasites), plant-pathogenic nematodes require a living host plant and cannot be cultured in artificial media. Nematodes must be carefully extracted from plant tissue or soil into water. Identified nematodes are then used to inoculate a healthy plant to recreate the original disease. Nematodes must be applied to the appropriate part of the plant, which is usually, but not always, the roots.

Soil samples are important in assays for both ectoparasites and endoparasites. In the case of ectoparasites, the soil may contain eggs, juveniles, and adults that would be lost if only washed roots were examined in the laboratory. In addition, even though endoparasites enter root tissue, soil samples may be used to monitor cysts, eggs, and infective juvenile stages.

It is important to consider where to sample for nematodes. Nematodes are often found in concentrated “hot spots” (or foci) in outdoor plantings. Nematode counts may be higher near the leading edge of a hot spot, where plants are still relatively healthy. Dead plants cannot support plant-pathogenic nematodes, so numbers may be low in soils where plants are severely affected.

In the absence of obvious root knots or cysts, soilborne nematodes must be diagnosed by assaying soil and root samples. Nematodes tend to congregate in “hot spots,” so a composite set of samples will most accurately reflect nematode populations. The time of year and where nematodes are expected in the soil profile must also be considered when samples are taken. After extraction, nematodes must be identified and counted under a microscope.

When affected plants are apparent, several soil samples should be obtained from both the affected area and from a nearby area that is apparently not affected. If nematode populations are high only in the affected area, it is likely that they are the cause of the plant symptoms. Similar nematode numbers in both affected and nonaffected areas suggest that nematodes are not the cause of the problem. Random samples should be taken when problem areas are not obvious. This maximizes the potential of recovering nematodes from areas of both high and low populations (Figures 4.16 and 4.17).

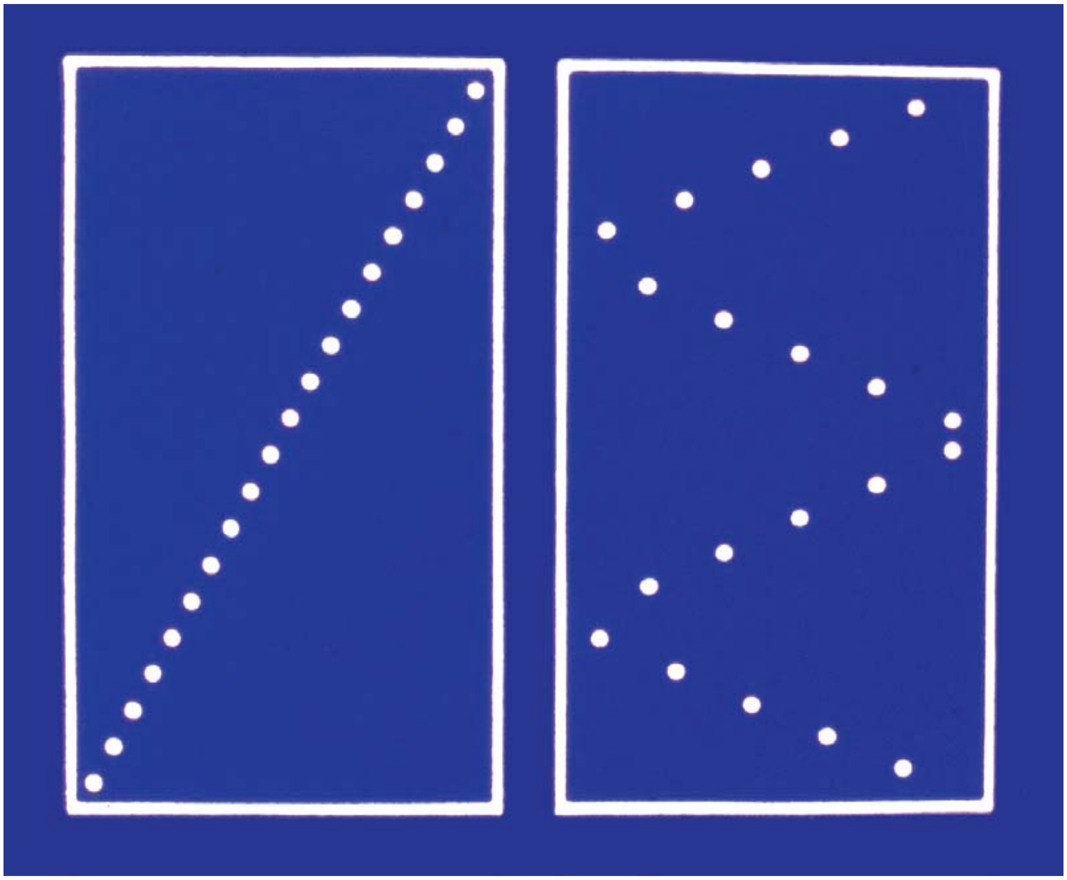


Figure 4.16. Sampling patterns for assessment of nematode populations.

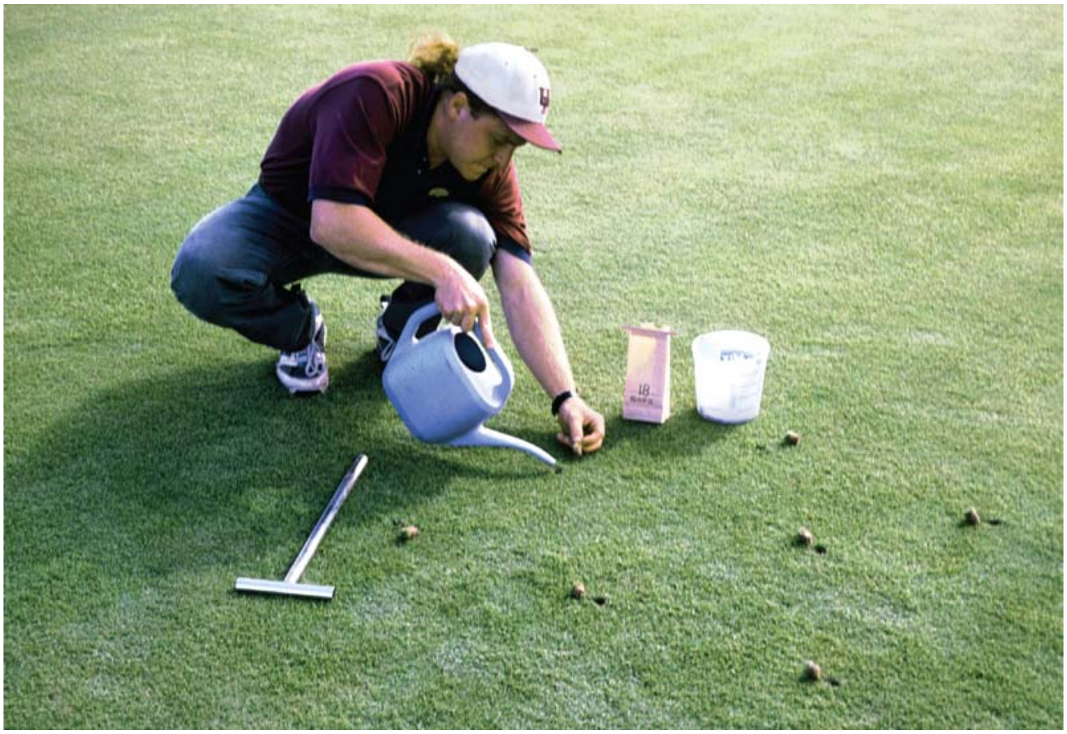


Figure 4.17. Sampling for nematodes on a golf putting green and filling the holes with sand.

Nematode populations can fluctuate over time, so the timing of sampling should be based on the recommendations of a diagnostic laboratory. For example, sampling from annual crops is usually recommended toward the end of the growing season when populations are often at their highest and enough time is available for the next spring's management decisions. Recommendations may vary, however, depending on the crop, the climate, and the type of nematodes expected to be present.

The depth of the soil sample can be important because different types of nematodes concentrate at different depths. In the absence of specific recommendations, multiple soil cores 10–20 centimeters (4–8 inches) deep should be combined to produce a sample. Soil and root samples should be kept moist and cool to keep the nematodes alive for identification. Saturating samples with water should be avoided to prevent drowning of the nematodes.

Plant-pathogenic nematodes may interact with other pathogens present in the soil. Sometimes they simply add to a plant's problems, but in other cases, **synergism** occurs (i.e., the plant exhibits symptoms that are more severe than expected), caused by the combination of pathogens. There have been many studies of synergism between species of nematodes, nematodes and other root pathogens, and nematodes and vascular wilt pathogens. For example, the **early dying** syndrome of potatoes involves lesion nematodes and Verticillium wilt, a fungal disease of xylem tissue. Some of these interactions are related to the wounds caused by nematode penetration and feeding, but nematodes can also predispose plants to other diseases by affecting their growth and physiology. For example, tobacco plants that were selected as resistant to black shank (a *Phytophthora* disease) became susceptible when the plants were first exposed to infection by root-knot nematodes.

How are nematode diseases managed?

Except in greenhouses and other artificial environments, one cannot expect to eliminate plant-pathogenic nematodes any more than one can eliminate all insects from a plant's environment. Plants may be infected by a variety of nematodes, and there are no specific management approaches for many nematodes, especially many of the ectoparasites.

Management choices for nematode problems are based on the damage potential of the specific nematode and the value of the plant. Once the nematodes are identified and counted, decisions may be based on the economic damage threshold (or **economic threshold**) for each type of nematode on various host plants. However, such thresholds often are not available.

Economic Damage Thresholds for Creeping Bentgrass Golf Putting Greens in the Northeastern United States

Nematode	Number (per 100 cubic centimeters of soil sample)
dagger	200
lance	400
needle	100
spiral	2,000
stubby-root	100
stunt	800

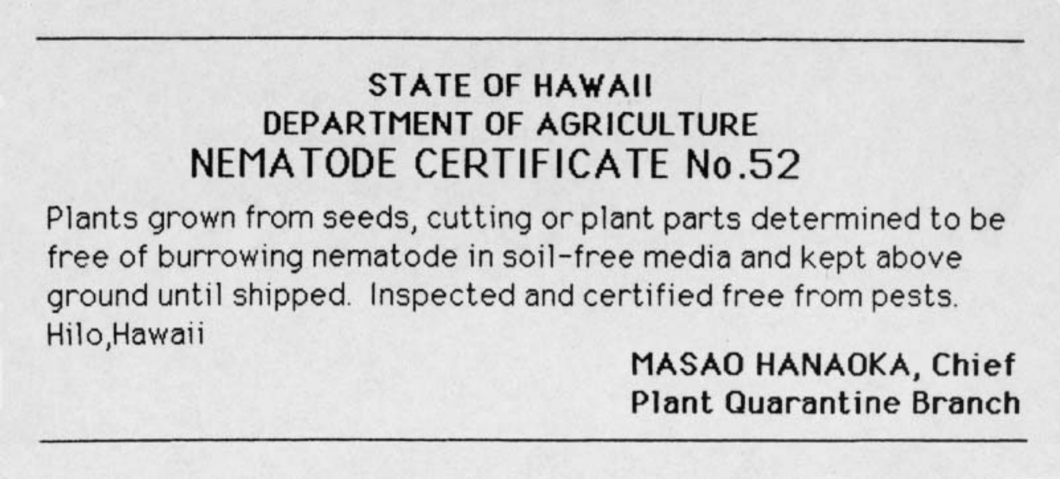
For annual crops, the majority of nematode management decisions should be made before planting. There are few cost-effective remedial treatments for nematodes once plants are infected. For perennial crops, good cultural practices, and, if necessary, a few nematicide treatments, may be sufficient to manage nematode problems. Severe disease levels in perennial plantings may require removal of infected plants and treatment of the infested area.

Avoidance

Healthy plants can tolerate reasonable populations of plant-pathogenic nematodes. Adequate water and fertilizer and optimal soil environments can help plants grow well despite the presence of nematodes. For example, when golf greens are mowed very short and water and fertilizer are minimized to create the desired hard surface for putting, nematodes can contribute to an overall decline of an already stressed turf. The same nematode populations may be of no consequence in higher cut lawns or athletic fields. Despite appropriate plant care, nematode populations may continue to increase in perennial plantings such as orchards, eventually surpassing economic damage thresholds.

Exclusion

Because nematodes are mostly soilborne pathogens, quarantines can help to delay the arrival of certain species in new areas. Nematode quarantines are in effect between and within countries. For example, forest products from the United States and Canada are restricted in Asia and Europe to avoid introduction of the pine wood nematode. Within the United States, certain citrus nematodes are found in only some of the citrus-growing states, and some nematodes in Hawaii are not present on the mainland. Movement of plants and soil between these areas is restricted (Figure 4.18). Similar restrictions are in effect to reduce the spread of the golden (cyst) nematode of potatoes out of the state of New York. Quarantine restrictions may require inspection of potted ornamental plants and rootstocks before shipments can be made. Soil and seeds contaminated with nematodes should be eliminated from planting materials to reduce the introduction of nematodes into new areas.



STATE OF HAWAII
DEPARTMENT OF AGRICULTURE
NEMATODE CERTIFICATE No.52

Plants grown from seeds, cutting or plant parts determined to be free of burrowing nematode in soil-free media and kept above ground until shipped. Inspected and certified free from pests.
Hilo,Hawaii

MASAO HANAOKA, Chief
Plant Quarantine Branch

Figure 4.18. A phytosanitary permit used as part of a program to exclude nematode pathogens. These certificates, issued by the country or state of origin, state that the plants are pathogen-free.

Quarantines can delay the spread of nematodes, especially those that are soilborne. Once nematodes are present, crop rotation and genetic resistance are common management approaches but are limited to certain types of nematodes. Heat treatment and chemicals can be used to kill all kinds of nematodes, but their use is restricted in certain areas. Optimal growing conditions help plants tolerate the presence of nematodes. Integrated management is most effective.

► DID YOU KNOW?

The golden (cyst) nematode of potatoes (*Globodera rostochiensis*) originated in South America and probably arrived in Europe in soil clinging to potato tubers. The nematodes were first discovered in the United States on Long Island, NY, in 1941. Machinery had been returned there in airplanes after World War I. The nematodes have spread to some additional farms in New York State, probably on farm machinery. Golden nematodes remain confined to nine counties within the state of New York because of the successful quarantine that has been in effect for more than 50 years.

Eradication

Removal of nematode-infested plant debris from the soil and exclusion of nematodes from irrigation water are key **sanitation** strategies for foliar nematodes in greenhouse operations.

Pine lumber and wood chips exported from North America must be treated with chemicals or heat to kill pine wood nematodes before these products will be accepted in Asia or Europe (Figure 4.19). Problems with stem and bulb nematodes can be reduced by elimination of obviously infected bulbs (showing necrosis or “nema wool”) at planting time. Sometimes infected bulbs are soaked in hot water or chemicals to eradicate the nematodes.

Figure 4.19. Lumber ready for kiln drying to eradicate pine wood nematodes before export to countries without this pathogen.

Most nematodes are quite sensitive to heat, so **steaming** of small quantities of soil, such as that in flower beds, will reduce nematode populations. **Solarization**, the use of plastic sheets to trap solar radiation and heat the soil, reduces nematode populations at shallow soil depths in sunny, warm climates. **Sanitation** of pots and benches with heat or chemicals (e.g., bleach) and **pasteurization** of planting media reduce nematode populations in greenhouses.

Even before the arrival of Europeans in the Western Hemisphere, the Incas of the Andes practiced fallow periods and **crop rotations** of 6–8 years for various crops, including potatoes, to improve yields. Potato cyst nematode populations decline 30–50% annually when a nonhost is grown and would probably never reach economically important population levels with 7-year rotations. Crop rotation is an important management tool for nematode problems of other annual plants if alternative nonhost crops can be grown economically. This is easier to implement for nematodes that are host specific, such as cyst nematodes. Rotation of corn and soybeans is a recommended practice to reduce populations of soybean cyst nematode (Figure 4.20).

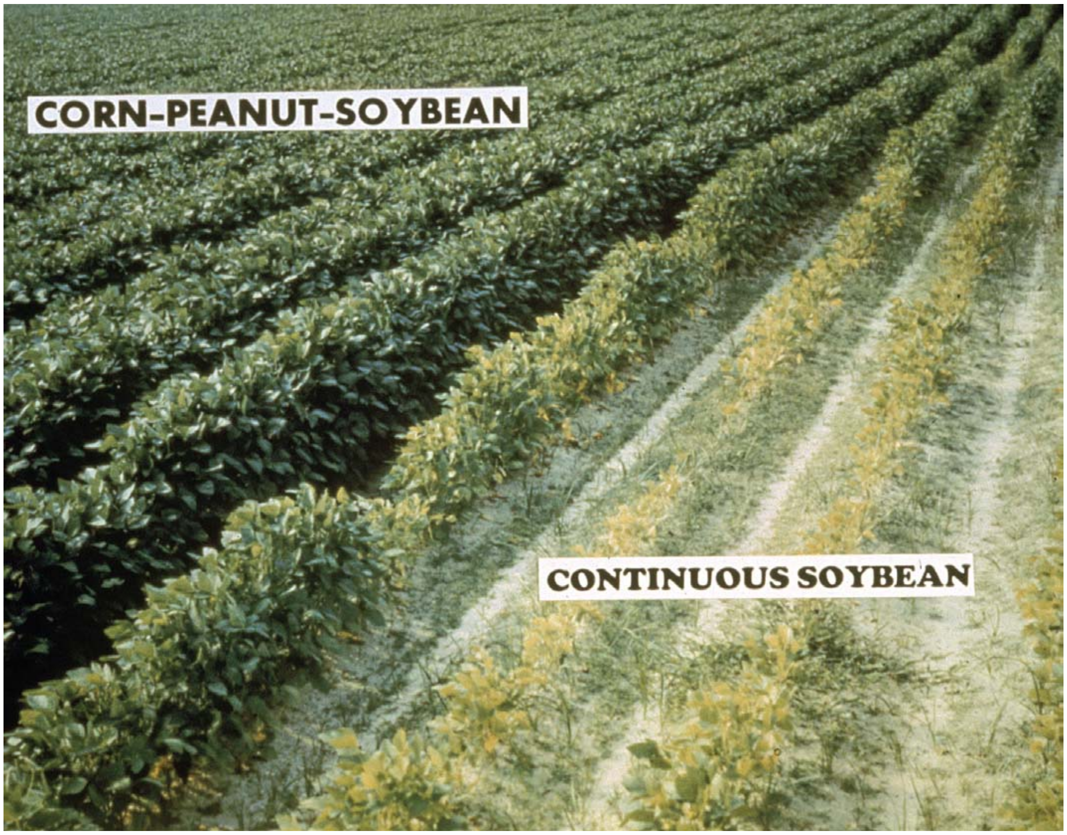


Figure 4.20. Field showing reduction in soybean cyst nematode damage to soybean in areas (left) where corn and peanut (nonhosts) were rotated with soybean. The area on the right was planted to soybean continuously.

There are fewer alternative crop choices for managing nematodes with wide host ranges, such as root-knot nematodes. These nematodes might survive and multiply on weed hosts even when their agronomic host has been temporarily removed.

When nematode populations exceed economic damage thresholds and other management practices are not sufficient, growers may consider the use of chemical (nematicide) applications to soil. Chemicals are effective against all kinds of nematodes, including the beneficial ones. Chemicals often are used before establishment or replanting of high value perennial plants such as fruit trees or strawberries. **Soil fumigants** harm growing plants but are sometimes used before planting to reduce the populations of nematodes as well as insect pests, fungi, and weeds (Figure 4.21). **Nonfumigant nematicides** can be applied to established plantings. These nerve poisons are toxic to nematodes, insects, and any living organisms with a nervous system, including humans. The toxicity and environmental hazards of nematicides and soil fumigants have led to severe restrictions to their use and bans on certain chemicals by the U.S. Environmental Protection Agency.

► DID YOU KNOW?

“Tear gas” (chloropicrin) is an effective soil fumigant used to manage nematodes in a number of crops.

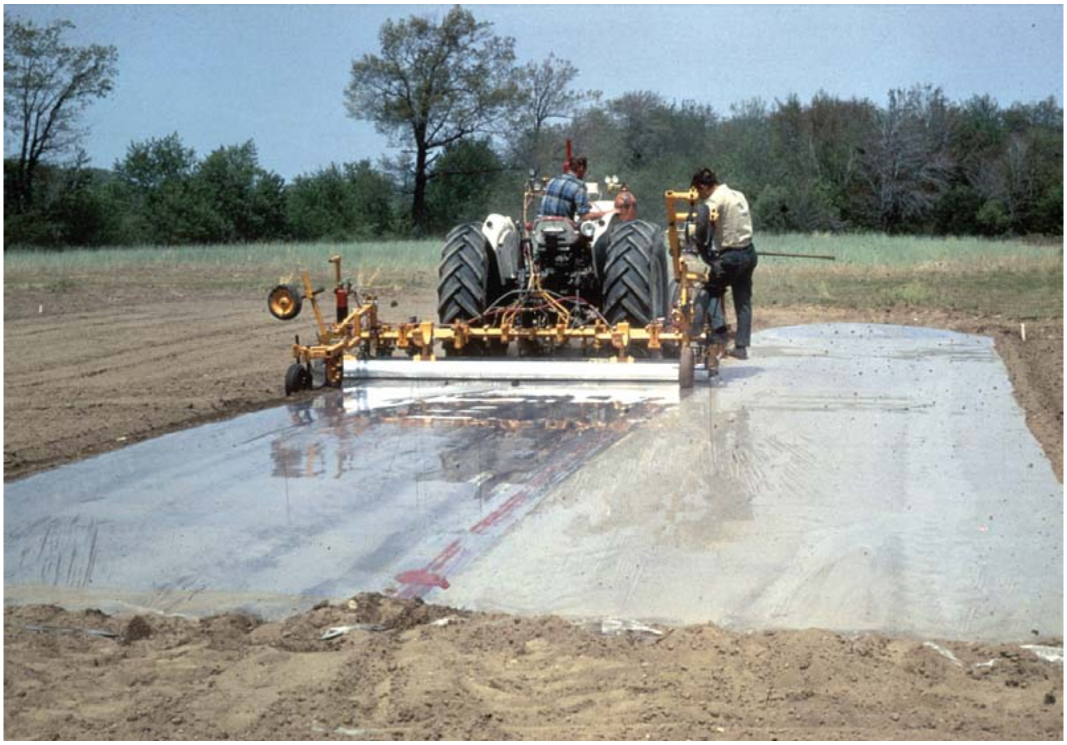


Figure 4.21. Application of methyl bromide, a soil fumigant, under plastic. Noncritical uses of methyl bromide were phased out in the United States in 2005 because it is considered a threat to the ozone layer. Certain allowances continue to be made for uses for which no technically and economically feasible alternatives have been identified.

Many soil fungi and other organisms are parasites or predators of nematodes and thus potential **biological control agents** (Figure 4.22). Successful use of these organisms in an economically viable system for the management of plant-pathogenic nematodes is still in the research stage. There also is ongoing research on compounds from plants that can kill or repel nematodes. Plant extracts, cover crops, companion crops, and green manures (in which living cover crops are plowed under before a susceptible crop is planted) are being studied.

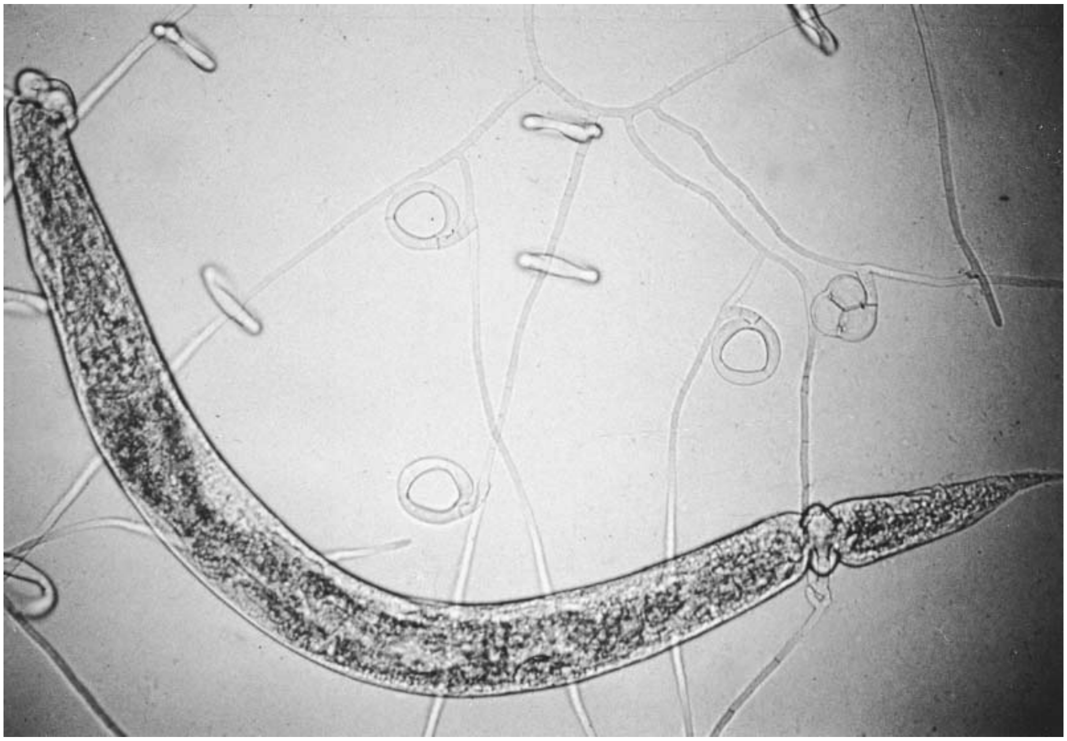


Figure 4.22. The fungus *Dactylaria brochopaga* traps nematodes by constricting a hyphal noose. It is one of many potential biological control agents for nematodes.

Protection

Foliar nematodes move onto plants in water films. In greenhouses, if water is applied only to the soil surface, leaf wetness is reduced, thereby reducing foliar nematode problems. Outdoors, where rain and dew may wet the leaves, mulch barriers can impede the movement of foliar nematodes from the soil to lower plant leaves.

Genetic resistance is most effective for nematodes that require specialized feeding sites. In resistant plants, a hypersensitive reaction creates dead cells near the feeding nematode, so it cannot establish its feeding site and dies. Resistance genes against root-knot nematodes or cyst nematodes are important management tools. However, continued use of these resistance genes can result in new biotypes of nematodes that can overcome the resistance. Careful monitoring of nematode populations is key to choosing resistant crop cultivars that provide the most protection (Figure 4.23).

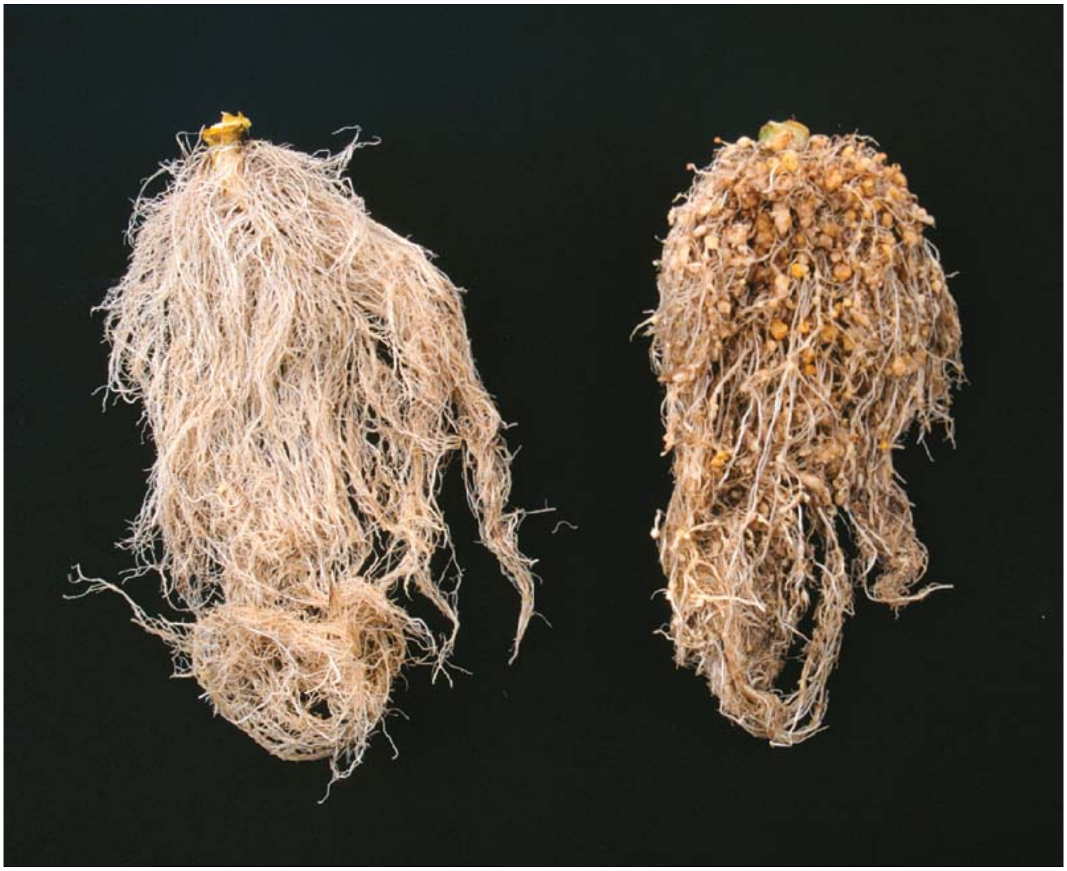


Figure 4.23. Roots of lettuce cultivar Salinas (left), resistant to root-knot nematodes, and of susceptible lettuce cultivar Ithaca (right).

Integrated management programs that use a variety of methods to reduce nematode populations are most effective. This is true for management of individual species of nematodes and for the more typical situation in which more than one species of nematode is present. Management choices should be based on soil and plant assays to determine whether nematode populations justify the cost and environmental impacts of any particular management strategy.

Management Strategies for Nematode Diseases

Avoidance

- Provide adequate water and fertilizer.

Exclusion

- Impose quarantines (local and international).
- Plant pathogen-free propagative parts.

Eradication

- Rotate to nonhosts.
- Remove weed hosts.
- Destroy infested plant debris.
- Use solarization.
- Steam soil.
- Fumigate soil.
- Apply nematicides.

Protection

- Plant resistant hosts.

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Questions

1. If you observe a plant with a disease caused by root-knot nematodes or lesion nematodes:
    a. What symptoms do you observe above ground for each?
    b. What symptoms do you observe below ground for each?
2. How can a plant-pathogenic nematode be distinguished from a free-living nematode?
3. What is the general life cycle of a plant-pathogenic nematode?
4. What are the common names for the following important plant-pathogenic nematode genera?
    Aphelenchoides
    Ditylenchus
    Heterodera
    Meloidogyne
    Pratylenchus
5. What kind of sample is needed to diagnose a nematode disease? Why should the results of a nematode assay include both the types of nematodes and their numbers per volume or weight of soil?
6. How could you differentiate between an angular leaf spot caused by a foliar nematode and one caused by a foliar bacterial pathogen?
7. Genetic resistance and rotation to a nonhost crop can be very effective for certain kinds of nematodes. Why are more general management practices also needed (steaming of soil, solarization, fumigation, nematicides)?
8. How could you manage pine wilt caused by the pine wood nematode? If you lose a pine tree to pine wilt and wish to replace it, what kind of pine is more likely to survive in that site?
9. Are nematode diseases more like monocyclic or polycyclic diseases, compared to diseases caused by other kinds of plant pathogens (e.g., fungi, bacteria)?

Challenge Questions

1. From the point of view of biology and management practices, are nematodes more similar to insect pests or to other plant pathogens (e.g., fungi, bacteria)?
2. If you were given a choice between selecting a cultivar of a new crop resistant to root-knot nematodes (*Meloidogyne* spp.) or resistant to lesion nematodes (*Pratylenchus* spp.), which project would most likely be successful and why?

Supplementary Resources

Illustrated Glossary

Special Topic

Introduction to plant-parasitic nematodes

Plant Disease Profiles

Burrowing nematode
Lesion nematode disease
Pine wilt disease
Root-knot nematodes
Soybean cyst nematode disease
Sting nematode

Case Study

Worms in the grass: A case study concerning the extraction and identification of plant-parasitic nematodes

► **WORDS TO KNOW**

The words in this list are defined in the Illustrated Glossary.

- avoidance
- biological control
- crop rotation
- cryptobiosis
- cuticle
- cyst
- economic threshold
- ectoparasite
- endoparasite
- eradication
- exclusion
- fumigant
- gall
- gene silencing
- giant cell
- juvenile
- knot
- migratory
- molt
- nematicide
- nematode
- parthenogenesis
- pasteurization
- protection
- RNA interference (RNAi)
- sanitation
- sedentary
- solarization
- stunting

stylet
syncytium (pl. syncytia)
synergism
vermiform
wilt

Exercise

Exercise 4.1. Match the terms and phrases with the images below. Use each term, phrase, and image only once.

Internet Resources

APS Resources

Alternatives to methyl-bromide: A Florida perspective
Effects of soil ecosystem management on nematode pests, nutrient cycling, and plant health
Plant nematological contributions to phytopathology

Recommended Websites

Nematode.net: Gene Sequencing Center for Parasitic Nematodes
The Nematode Song Book
Plant and Insect Parasitic Nematodes
(An outstanding collection of information ranging from photo galleries to molecular diagnostics for plant- and insect-parasitic nematodes)
Worm Book: Online review of *C. elegans* biology

Internet Research

Internet Research 4.1. Find a fact sheet or disease profile for soybean cyst nematode (*Heterodera glycines*). Are resistant soybean cultivars recommended? Find a fact sheet or disease profile for foliar nematode (*Aphelenchoides ritzemabozi*) of chysanthemums. Are any resistant cultivars recommended? What other management practices are recommended for each disease? Explain the management differences between these two nematode problems.

Internet Research 4.2. Choose a plant of interest and find a fact sheet or disease profile that describes the kinds of nematodes that are important pathogens of this plant. Find recommendations on sampling for nematodes on this plant and what economic damage thresholds are available, especially in your geographic area. Where can you obtain a nematode assay near you?

CHAPTER 5

What Are the Causes of Plant Diseases?

► Viruses

IN THIS CHAPTER:

- ► **What are viruses?**
- ► **What symptoms and signs do viruses cause?**
- ► **How do viruses survive and spread?**
- ► **How are virus diseases diagnosed?**
- ► **How are virus diseases managed?**

What are viruses?

Viruses have been known for over 100 years (Figure 5.1), and for nearly that long scientists have debated whether or not these relatively simple pathogens are living or nonliving. Viruses are infectious and intracellular. They cannot reproduce on their own and require components found in the cells of their hosts. However, viruses do organize their own reproduction within host cells and can cause disease. Several theories have been proposed to explain the origin of viruses. Some virologists believe that viruses are the descendants of genes that "escaped" from host cells; others believe that viruses evolved as independent life forms, likely predating other forms of life.

Characteristics of Viruses

- particle dimensions ranging from 20 to 2,000 nanometers
- genome composed of nucleic acid
- capsids composed of protein
- ability to replicate within host cells
- ability to cause disease, in most cases

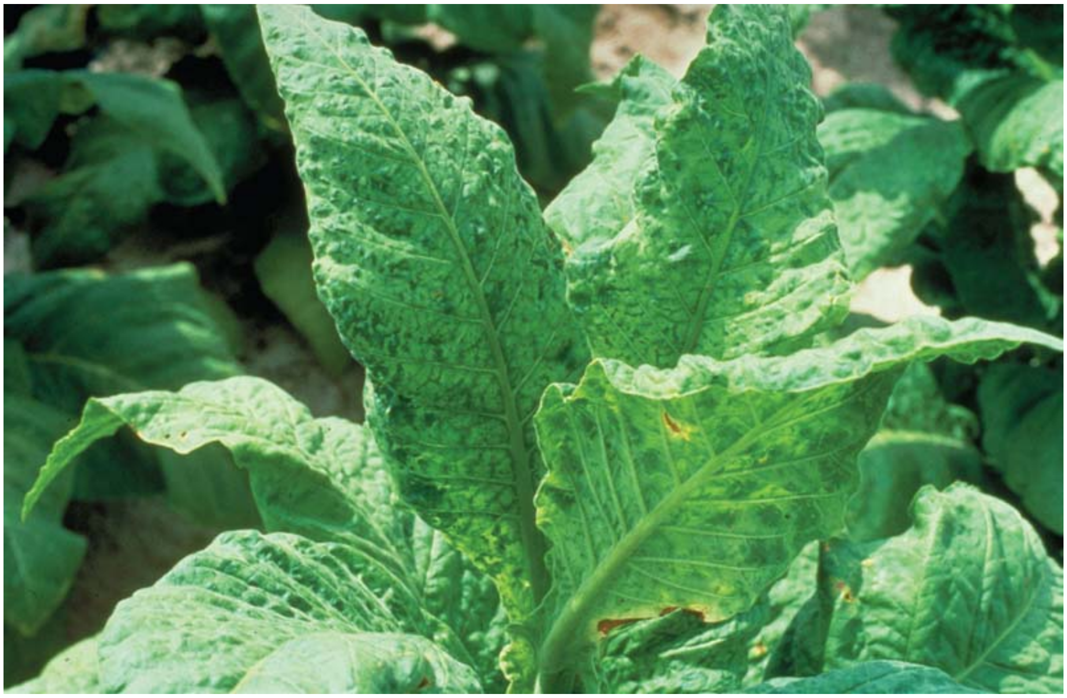

Figure 5.1. Tobacco leaves with symptoms caused by *Tobacco mosaic virus* (TMV). Scientific studies of TMV began in the 1890s, and the virus has been used since then as a model in the study of viruses and their interactions with host plants.

Viruses are known to infect bacteria, algae, fungi, plants, and animals, including people. Many human illnesses, from the common cold to the life-threatening AIDS, are caused by viruses. Since the concept of a virus was first described by the Dutch scientist Martinus Willem Beijerinck (Figure 5.2) in 1898, close to 1,000 species of viruses that infect plants have been described. Each of these species consists of many slightly different variants, which are designated as **strains** once they have been studied and characterized.

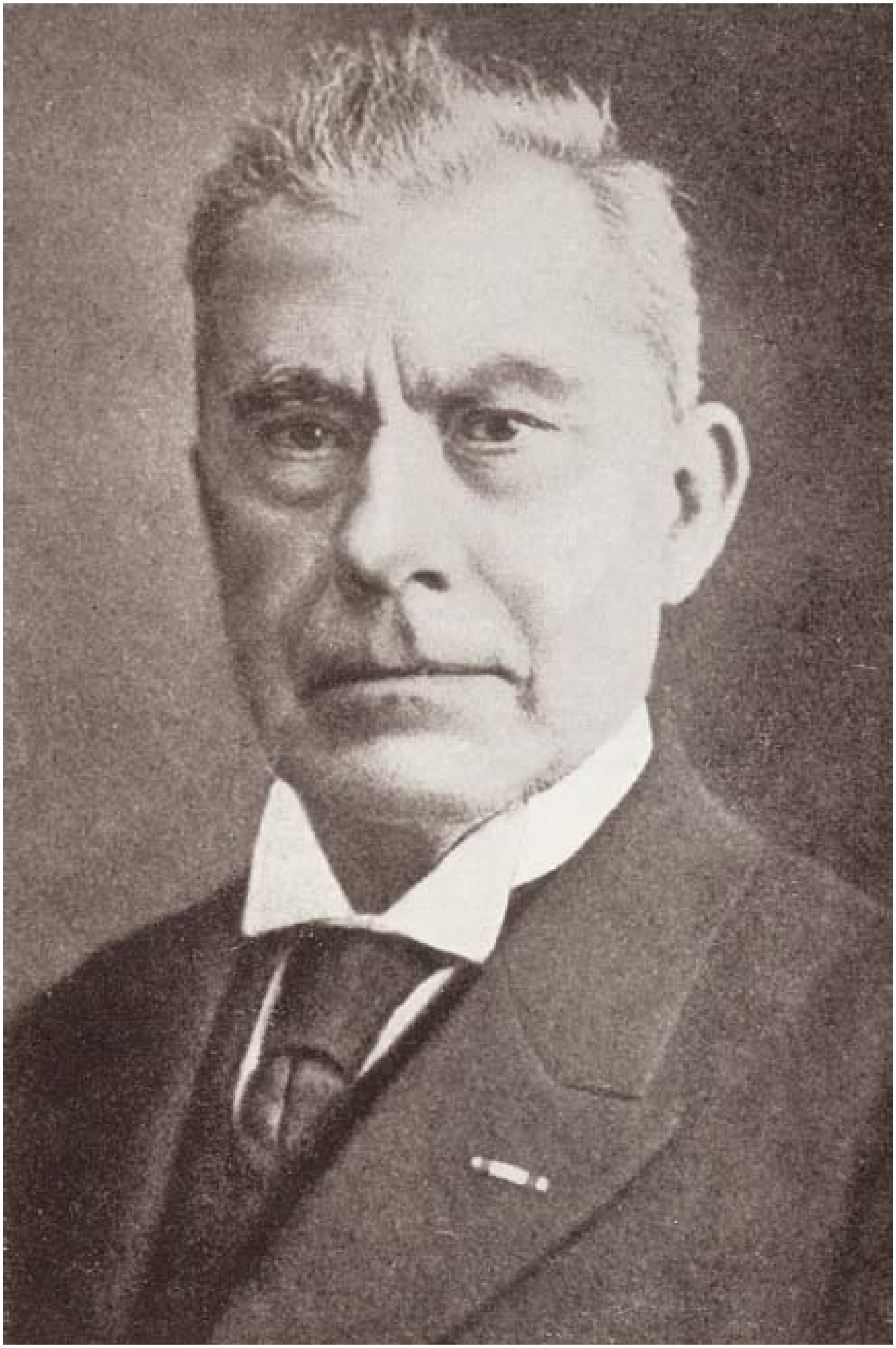

Figure 5.2. Martinus Willem Beijerinck (1851–1931) was the first scientist to propose that what we now call viruses are fundamentally different from other disease-causing agents, principally bacteria. His concept of a

“*contagium vivum fluidum*” (a contagious living fluid) was groundbreaking, and for this insight he has been called the “father of virology.”

Individual virus particles, or **virions**, are much too small to be seen with the naked eye or even with the aid of a light microscope. They are measured in **nanometers** (1 nanometer = 1 nm = 10^{-9} meter). Particles of different viruses have characteristic shapes. Some are roughly spherical or **icosahedral**, and some are **rods**, which can be either **rigid** or **flexuous** (Figure 5.3). The smallest spherical viruses are less than 20 nm in diameter, whereas the largest rod-shaped viruses are over 2,000 nm long. In comparison, a typical bacterial cell is 1,000 × 3,000 nm. Even the longest rod-shaped plant viruses are not visible to the naked eye because their diameter (typically less than 15 nm) is smaller than the wavelength of light. Some plant viruses have other shapes, such as the bacilliform viruses (shaped like *Bacillus* bacteria, but much smaller) in the family *Rhabdoviridae* and the twin particles of viruses in the family *Geminiviridae*. The different sizes and shapes of plant viruses are useful for both classification and diagnosis.

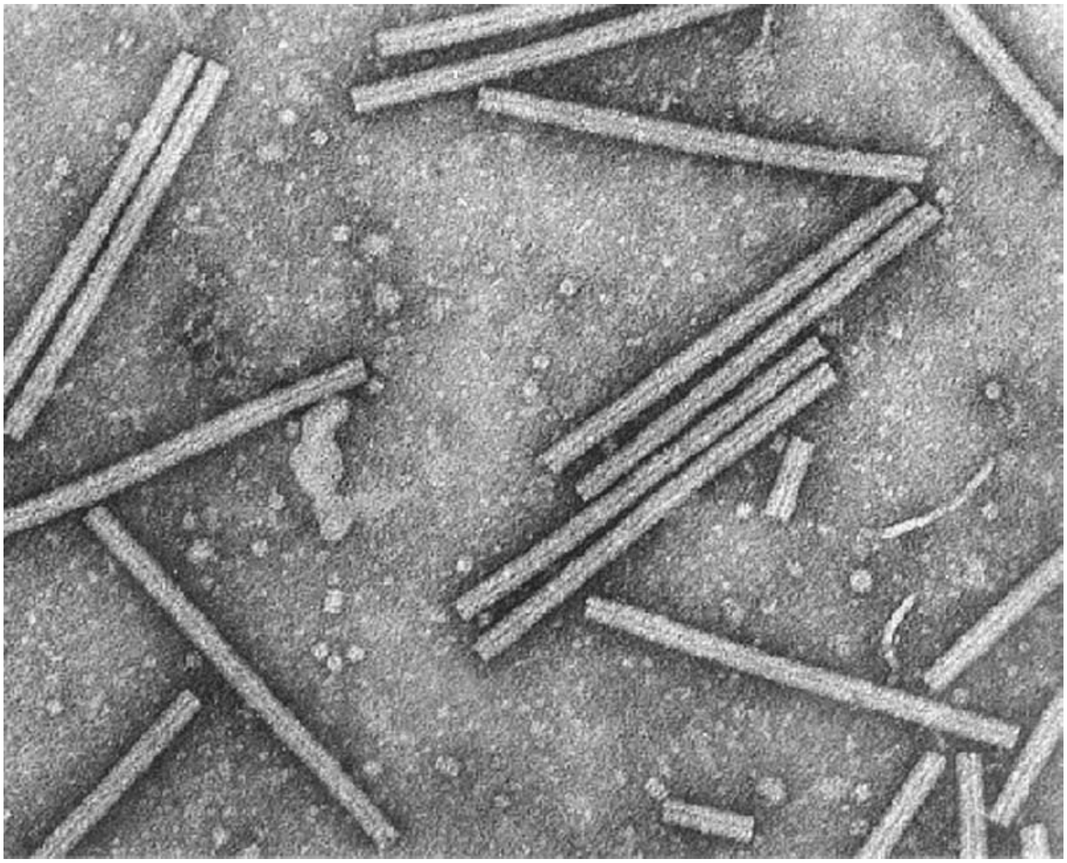

Figure 5.3. Rod-shaped particles (300 × 15 nm) of *Tobacco mosaic virus*, in an electron micrograph.

Most of the viruses that infect plants are composed of only two types of macromolecules: a nucleic acid genome and one or more proteins that form a coat, or **capsid**, which encloses or **encapsidates** the nucleic acid. The viral capsid is composed of many copies of usually one or sometimes a few small proteins encoded by the viral genome. The genome is composed of either **ribonucleic acid (RNA)** or **deoxyribonucleic acid (DNA)**, which can be either single-stranded (**ss**) or double-stranded (**ds**) (Figure 5.4). Most plant viruses have ssRNA genomes, which either directly encode proteins (**positive sense**, or **+sense**) or have proteins encoded on the complementary (mirror-image) strand (**negative sense**, or **–sense**). For example, the genome of the first plant virus described, *Tobacco mosaic virus* (TMV), is +sense ssRNA. The TMV genome consists of about 6,400 nucleotides, encapsidated by 2,130 copies of the TMV coat protein, forming a rigid, rod-shaped (300 × 15 nm) virion (Figure 5.5). The genomes of about two-thirds of all plant viruses are composed of +sense ssRNA, like that of TMV.

► DID YOU KNOW?

Wendell M. Stanley (1904–1971) received a Nobel Prize for his research on the crystallization of *Tobacco mosaic virus* (TMV) in 1935. This groundbreaking work, published in the elite journal *Science*, actually contained a significant oversight. Stanley concluded that TMV was composed solely of protein when he chose to ignore the small amount of phosphorus (from the viral RNA) found in his assays. It was left to Fredrick Bawdin and Norman Pirie in 1936 to show that virus particles consist of nucleic acid and protein. The importance of viral nucleic acid wasn't demonstrated until the 1950s.

► DID YOU KNOW?

Most plant viruses are named for the first plant in which they were found (or an economically important host) and a symptom they induce. For example, *Tobacco mosaic virus* was first identified in tobacco (*Nicotiana tabacum*), in which it causes a yellow and green mosaic. Closely related viruses are grouped into a genus, which is often named for a key member of the group (e.g., *Tobamovirus*). Virus names are also abbreviated as acronyms, created from the first few letters of their names (e.g., TMV for *Tobacco mosaic virus*). Virologists are not yet in agreement on the best scheme for formally naming viruses.

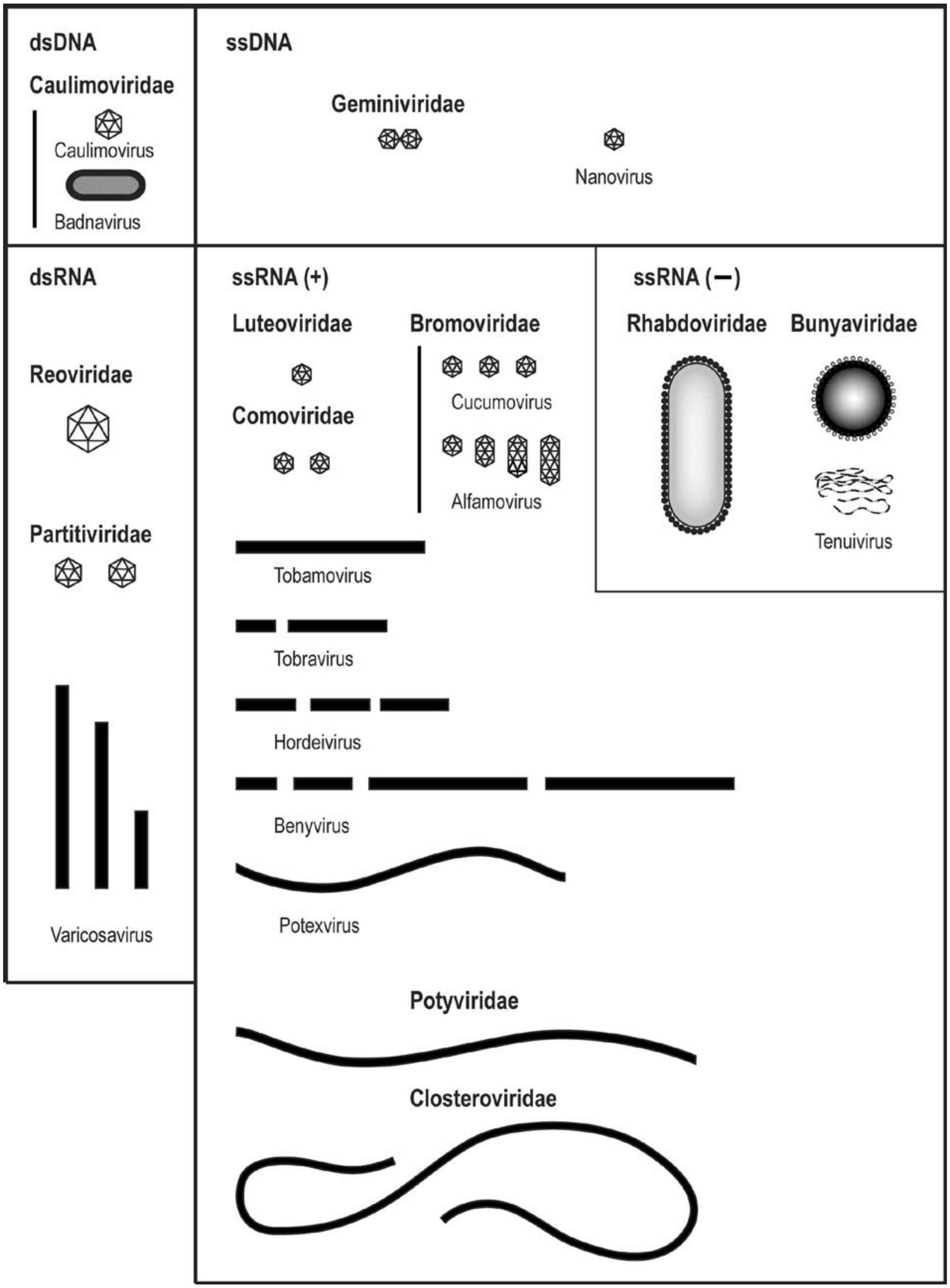
dsDNA
Caulimoviridae
Caulimovirus
Badnavirus
ssDNA
Geminiviridae
Nanovirus
dsRNA
Reoviridae
Partitiviridae
Varicosavirus
ssRNA (+)
Luteoviridae
Comoviridae
Bromoviridae
Cucumovirus
Alfamovirus
Tobamovirus
Tobravirus
Hordeivirus
Benyvirus
Potexvirus
Potyviridae
Closteroviridae
ssRNA (−)
Rhabdoviridae
Bunyaviridae
Tenuivirus

Figure 5.4. Some of the families and genera of plant-pathogenic viruses. Note the variety of shapes of virus particles and the different types of nucleic acid genomes.

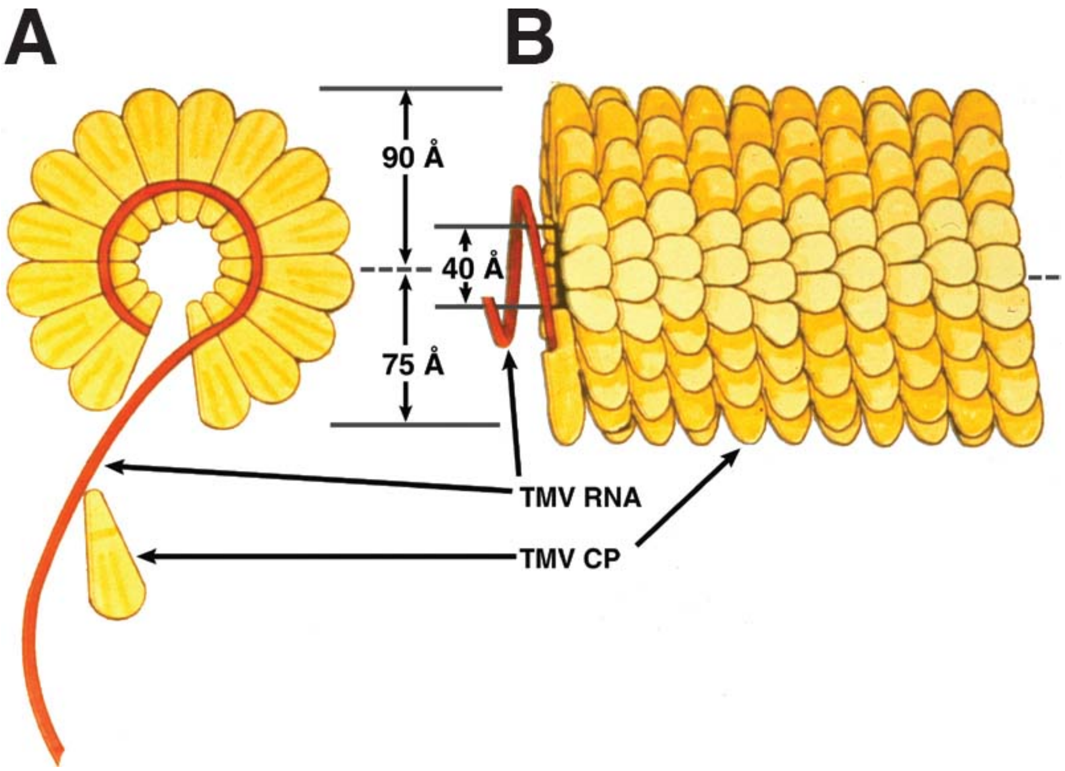

Figure 5.5. Structure of a *Tobacco mosaic virus* (TMV) particle. The virus particle is made up of a spiral or helix of 2,130 coat protein (CP) subunits and a single-stranded positive-sense RNA of approximately 6,400 nucleotides. The relationship between the CP subunits and the single-stranded RNA is shown in a transverse view (A) and a longitudinal view (B) (10 Å = 1 nm).

All viruses are obligate parasites. They cannot reproduce outside a living host cell, because their reproductive process, called **replication**, requires amino acids, nucleotides, enzymes, cellular structures such as ribosomes and membranes, and energy supplied by the host cell.

The genomes of plant viruses are very small, containing as few as four genes, also called **open reading frames** (**ORFs**). In some viruses, the genes are located on a single piece of nucleic acid. Other viruses have **multipartite** genomes, in which the genes are located on more than one piece of nucleic acid. In some multipartite viruses, the separate pieces of nucleic acid are enclosed in a single virus particle. Other multipartite viruses are **multiparticulate**, with pieces of the nucleic acid genome enclosed in more than one particle. Usually, all parts of the genome must be in the same host cell at the same time for infection and replication to occur.

Multipartite Genomes and Multiparticulate Viruses

- The genome of *Tobacco mosaic virus* (genus *Tobamovirus*) contains four genes (open reading frames) located on a single piece of RNA.
- The genome of viruses in the genus *Tospovirus* (family *Bunyaviridae*), such as *Tomato spotted wilt virus*, consists of three pieces of RNA enclosed within a single virus particle. Viruses in which genes are located on separate pieces of nucleic acid have a **multipartite** genome.
- The genome of viruses in the family *Bromoviridae*, such as *Cucumber mosaic virus*, consists of three pieces of RNA encapsidated in different particles. Viruses in which the genes are located on separate pieces of nucleic acid contained in different particles are called **multiparticulate**. Viruses in the family *Bromoviridae* are both multipartite and multiparticulate.

When a virus infects a plant cell, the genome of the virus must be made available to be copied to produce new viral genomes (**replication**) and to be used for the production of virus-encoded proteins (**translation**). The term *replication* is used to refer to both the copying of the viral genome and the reproduction of entire virions because these two processes are so tightly coupled. Both nucleic acid replication and protein translation require cellular structures (e.g., ribosomes) and molecules provided by the host cell.

In +sense ssRNA viruses, the genome itself is a **messenger RNA** (**mRNA**) and has genes that encode some of the proteins required for virus replication (Figure 5.6). Some of these proteins are enzymes that are required for virus replication. One early product of the translation of the viral RNA is part or all of the enzyme complex required to copy the genome, an **RNA-dependent RNA polymerase**, or **replicase**. Two of the four genes on TMV RNA encode proteins that form the virus replicase complex. This enzyme complex, along with host components, first produces complementary copies (–sense RNA) of the viral RNA.

General Steps in the Replication of a +sense ssRNA Virus

- coat protein removed
- replicase produced
- complementary –sense RNAs produced
- multiple new copies of +sense RNAs produced
- movement protein produced
- coat protein produced
- new virions assembled

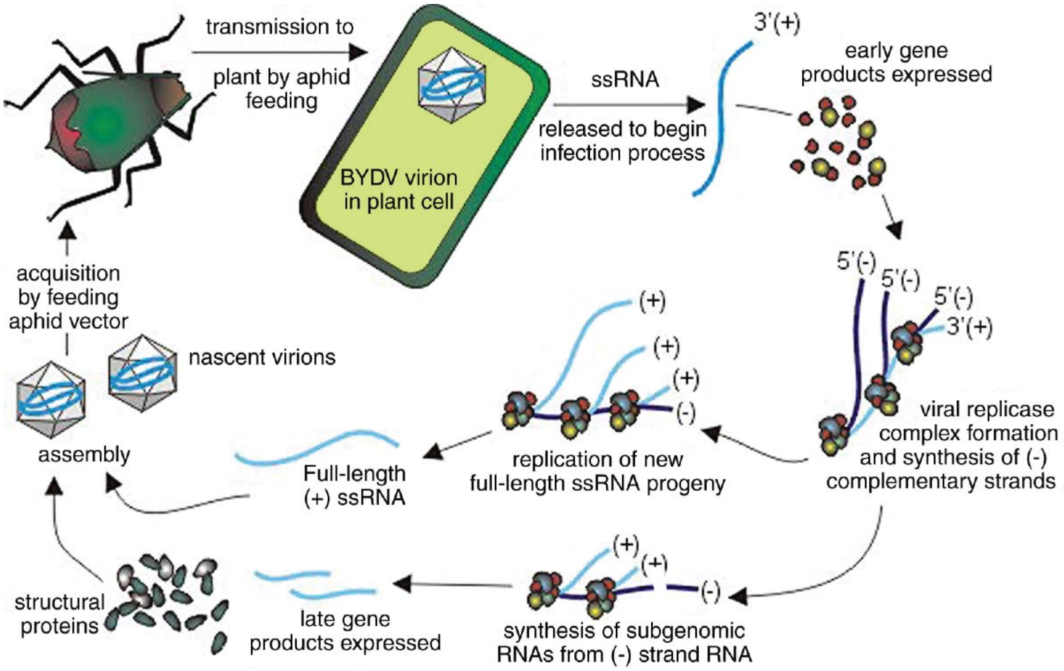
transmission to
plant by aphid
feeding
BYDV virion
in plant cell
ssRNA
released to begin
infection process
3'(+)
early gene
products expressed
5'(-)
5'(-)
5'(-)
3'(+)
viral replicase
complex formation
and synthesis of (-)
complementary strands
(+)
(+)
(+)
(-)
replication of new
full-length ssRNA progeny
Full-length
(+) ssRNA
acquisition
by feeding
aphid vector
nascent virions
assembly
(+)
(+)
(-)
synthesis of subgenomic
RNAs from (-) strand RNA
late gene
products expressed
structural
proteins

Figure 5.6. Replication cycle of *Barley yellow dwarf virus* (BYDV), a +sense ssRNA virus.

The –sense RNAs are used to make multiple copies of the original +sense viral RNA (Figure 5.6). Thousands of copies of +sense viral RNA can be produced in a single infected host cell. Other plant viruses with –sense ssRNA, dsRNA, ssDNA, or dsDNA genomes replicate their nucleic acids through a variety of different strategies.

Genes (or ORFs) on newly formed genome copies can be translated later to produce additional protein products. For example, two proteins are produced later in TMV replication. **Movement protein** facilitates viral RNA movement from cell to cell in the plant through the **plasmodesmata**, the cytoplasmic strands that connect plant cells. **Coat protein** encapsidates the new viral RNAs to form virions, hundreds to millions of which can be produced in each infected host cell. The assembly of plant viruses is the final step of replication. For some viruses, assembly requires no host components. Several plant viruses have been assembled by scientists in the laboratory by mixing purified viral coat proteins and nucleic acids under the proper conditions. Other viruses require plant proteins or membrane structures for assembly and thus cannot be assembled in vitro.

Typically, the mRNAs of eukaryotic organisms such as plants and animals contain a single ORF and encode a single protein. Viruses that multiply within eukaryotic cells have evolved strategies to express multiple, functionally distinct proteins from a single viral RNA molecule with more than one ORF. Some TMV ORFs, for example, are expressed from smaller pieces of RNA produced during replication, called **subgenomic RNAs**. In contrast, in the replication of *Tulip breaking virus*, the entire viral genome is translated into one large **polyprotein**, which is then cleaved by proteases (protein-degrading enzymes) into the active protein products.

Variability in a population of viruses arises mainly through mutations, or errors, in the nucleotide base sequence that occur during replication. DNA copying is a relatively error-free process (one error in 10^8 nucleotides), but RNA copying is much less reliable (one error in 10^4 nucleotides). Viruses with RNA genomes are subject to this high error rate, which leads to variability in the population and to the evolution of new virus strains and species.

A virus is an infectious, submicroscopic, intracellular agent, composed minimally of nucleic acid and protein, that directs its own reproduction in living host cells only and is capable of causing disease. Most plant viruses have an ssRNA genome.

► **DID YOU KNOW?**

Post-transcriptional gene silencing (PTGS) (see Chapter 9) is a host plant defense in which viral RNAs created during replication are selectively degraded. Plant viruses have evolved ways to suppress or evade PTGS so that they can successfully infect a host. Suppression of PTSG results in symptoms, including reduced quality and yield.

What symptoms and signs do viruses cause?

Viruses cause a range of symptoms (Figure 5.7), including some that are highly diagnostic and others that can easily be confused with symptoms of other biotic or abiotic agents. Many diseases caused by phytoplasmas (see Chapter 3) were originally thought to be caused by viruses, because of their similar symptomatology. Some viruses do not produce symptoms in some hosts, and one group of viruses causes no obvious symptoms in most of its hosts. Most symptoms caused by viruses are **systemic** rather than localized. The development of **local lesions** (small necrotic or chlorotic spots at the site of virus infection in some host plants) has been used extensively by virologists to study the mechanisms of virus infection and replication.

► DID YOU KNOW?

Following the work on *Tobacco mosaic virus* at the end of the 1800s, many mosaic diseases were described. However, since the causal viruses were invisible and could not be isolated for study, there was considerable confusion about these diseases and their causal agents until the 1930s. Some scientists thought that all mosaic diseases were caused by a single entity and that it would be possible to manage them all in the same way. If only it were so simple!

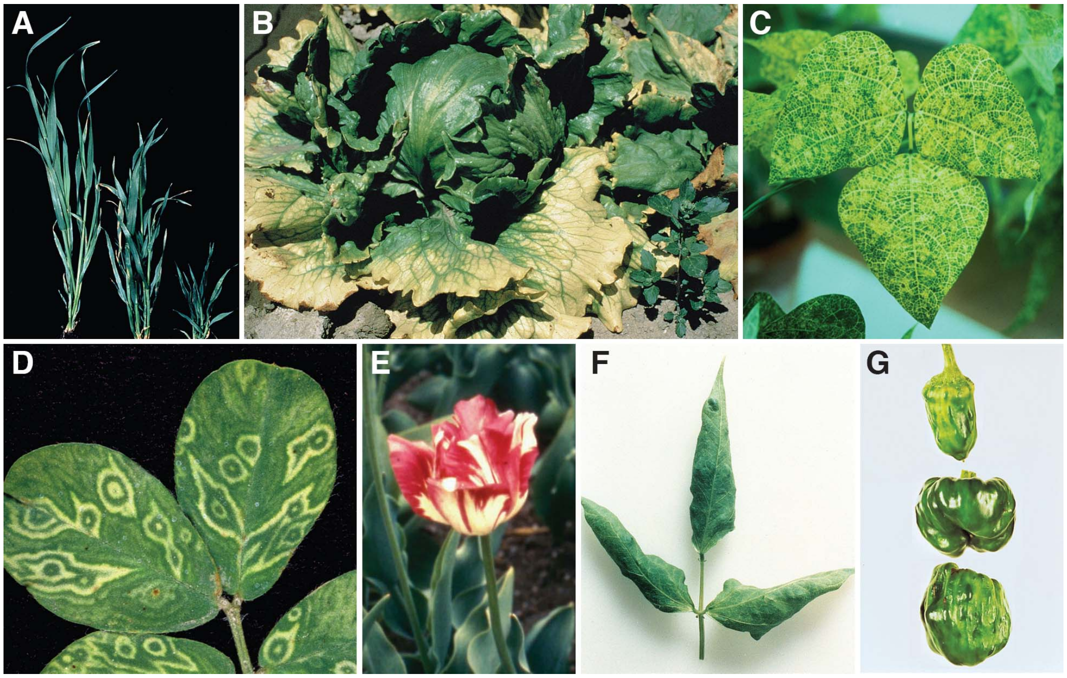

Figure 5.7. Common symptoms caused by viruses. A, Stunting or dwarfing (a healthy plant is on the left). B, Yellows. C, Mosaic. D, Ringspot. E,

Flower break. F, Leafroll. G, Fruit deformation.

The most common symptoms of plant virus infection (Figure 5.7) are **stunting** or **dwarfing, chlorosis** (yellowing), and other discolorations and malformations. Chlorosis can be general (in **yellows** diseases) or can develop in patterns, such as **mosaics** or **mottles** of yellow and green patches, **ringspots** of roughly circular chlorotic and green tissue, or **vein banding** of yellow areas along leaf veins. Patterns of discoloration also can occur on flowers (**flower break**) and fruits. Chlorosis reduces the photosynthetic area of the plant, which reduces photosynthate production and thus indirectly contributes to the stunting or dwarfing of the plant.

DISEASE CLASSIC

Tobacco Mosaic

Hosts tobacco, tomato, and other solanaceous plants
Pathogen *Tobacco mosaic virus* (TMV)

Symptoms Mosaic, mottling, necrosis, stunting, leaf curling, and yellowing of plant tissues can occur, depending on the host. Symptoms depend on the age of the infected plant, environmental conditions, virus strain, and host genotype. In tomato, the fruit are often distorted and nonuniform in color, and yield is reduced.

Disease Cycle TMV is easily transmitted when an infected leaf rubs against a healthy leaf or when plants are touched with contaminated tools or hands. The virus can also be present on the surface of seeds. A microscopic wound is required for TMV to enter a plant cell, where it replicates and produces hundreds to thousands of new virus particles. The virus spreads within the infected plant from cell to cell via plasmodesmata and over long distances via the vascular system.

Management Contaminated seed, tools, and hands should be treated to destroy the virus. Contaminated soil and infected seedlings should be discarded. During the growing season, infected plants should be rogued and destroyed. Several tobacco and tomato cultivars have genetic resistance to TMV.

Significance TMV was used for many "firsts" in the field of plant virology. It was the first virus to be recognized as different from other disease-causing agents, the first virus to be purified, and the first to be seen with an electron microscope. TMV was the first virus shown to consist of nucleic acid and protein and the first in which it was demonstrated that the nucleic acid alone is infectious. The TMV genome was the first to be completely sequenced and the first used to create a plant genetically modified to resist virus infection.

Plant Disease Profiles: Tobacco mosaic

Plants that are infected with a virus are often stunted or deformed and have either localized or extensive areas of chlorosis. Mosaic and ringspot symptoms are often diagnostic of a virus infection, but other virus-induced symptoms are easily confused with those caused by other abiotic and biotic agents.

► **DID YOU KNOW?**

Color-breaking viruses can cause unexpectedly beautiful symptoms in flowers. In 17th-century Holland, tulips were a status symbol, and "broken" tulips, with streaks of bright colors and white, were especially prized. The "tulipomania" craze occurred

> when speculators drove prices of these bulbs to extraordinary levels. In 1636, a single diseased bulb was traded for 4 tons of wheat, 8 tons of rye, 2 tons of butter, 1,000 pounds of cheese, 4 tons of beer, 2 barrels of wine, four oxen, eight swine, 12 sheep, a suit of clothes, a bed, and a silver drinking cup! In 1637, inevitably, the bubble burst and the tulip market collapsed, dragging the entire Dutch economy down with it.

Viruses rarely kill their hosts or cause extensive **necrosis**. Necrosis is sometimes visible in leaf veins of virus-infected plants. Since viruses infect living cells, they are usually found in the phloem rather than in the nonliving xylem; thus, wilting is not a common symptom. One relatively common necrotic symptom of virus infection is **phloem necrosis**, which results from degeneration of the vascular tissue when cells are killed by virus infection. Phloem necrosis causes a disruption in the flow of photosynthates within the plant, which contributes to stunting.

Malformations caused by virus infection can be seen in different parts of plants, including leaves, stems, and fruits that develop abnormal shapes. Examples include **rugosity** (leaf puckering caused by retarded vein growth), **leaf rolling** at the tips or edges, **stem pitting** or grooving, and **enations** or **tumors** resulting from abnormal proliferation of plant tissues. These symptoms suggest that virus infection interferes with gene expression in the host plant.

It is impossible to see individual virions with the naked eye or a light microscope, but crystalline clusters of viruses and viral products called **inclusion bodies** are sometimes visible with a light microscope. Experience is required to recognize these signs of virus infection and to use them for tentative disease diagnosis.

Individual virions and the changes that they induce within plant cells (their **cytopathology**) can be visualized with an **electron microscope**. The sizes and shapes of virions, as well as some distinctive cytopathological changes, can be useful clues in diagnosis of a virus disease.

How do viruses survive and spread?

Viruses produce no specialized survival structures, as do many fungi and some nematodes. They are essentially inanimate molecules when they are not in a living host cell. Some plant viruses can survive on tools, in soil, or in plant debris as virions that must be introduced into a living cell to reproduce and cause disease. How long they can survive depends on the particular virus. Some viruses, such as TMV, are quite stable and can survive for years, but many others are destroyed in hours or days outside living host tissue. Unstable viruses typically survive within plant structures, such as seeds or tubers, or within **vectors**. Vectors are organisms, mostly insects, that transmit viruses.

Viruses Are Transmitted from Plant to Plant by

- insects
- nematodes
- mites
- sap (mechanical transmission)
- fungi and fungus-like organisms

To be perpetuated, plant viruses must be transmitted both from plant to plant within a population and from one generation to the next. Most plant-to-plant virus transmission within a population requires a healable **wound**, such as is created by an insect or nematode stylet. If a virion is simply deposited on a leaf surface or is present in the soil on or near the roots of a plant, it has no means of moving and crossing the cuticle, cell wall, and cell membrane. Viruses can be spread from plant to plant by **mechanical transmission** (**sap transmission**), in which viruses in sap are passed from an infected plant to a healthy plant that has a wound through which viruses can enter. This can occur when the wind whips the leaves of neighboring plants against each other or when people injure plants with machinery or tools. In nature, only a few viruses are spread from plant to plant by mechanical transmission. However, mechanical transmission is an important experimental tool for virologists, who inoculate plants by rubbing leaves with plant sap containing viruses. Tiny, abrasive particles are applied to the leaves to create microscopic wounds for virus entry during inoculation.

In nature, vectors, most often insects, create the wounds that permit plant-to-plant transmission of plant viruses. Insect transmission is usually a very specific process that requires recognition of specific viral proteins by specific proteins in the insect vectors. Examples of these recognition proteins are the helper proteins produced by viruses such as *Tulip breaking virus*.

Insect transmission of plant viruses has been recognized for over 100 years. The most important insect vectors are those that have slender **piercing-sucking** mouthparts, called **stylets**, which allow them to pierce and feed on plant cells without always killing the cells (Figure 5.8). These insects can acquire viruses from cells of infected plants as they feed and later introduce them into cells of healthy plants via their saliva during feeding.

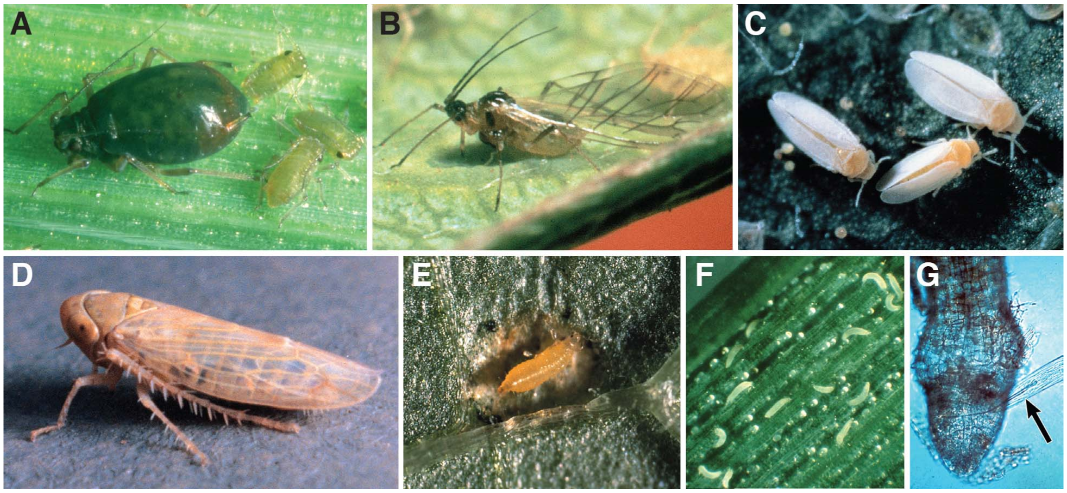

Figure 5.8. Vectors of plant viruses. A, Wingless (apterous) aphids. B, Winged (alate) aphid. C, Whiteflies. D, Leafhopper. E, Thrips (larva). F, Eriophyid mites. G, Migratory ectoparasitic nematode (arrow) feeding at a root tip.

► DID YOU KNOW?

Geminiviruses were first described in the mid-1970s. By the 1990s, as a result of the global movement of plants and whiteflies that transmit these viruses, geminiviruses had become major pathogens of vegetables and cotton in the subtropics and tropics in the Western Hemisphere. Similarly, tospoviruses were of limited significance until the mid-1980s. With the emergence of the western flower thrips vector as a worldwide resident and with worldwide shipping of plant materials, tospoviruses have become a global problem, affecting more than 650 plant species, including peanuts, flower crops, and many vegetables.

Each plant virus is typically transmitted by only one kind of insect vector, although it may be transmitted by many different species of that vector. The most common insect vectors of viruses are in the order Hemiptera, including aphids, leafhoppers, planthoppers, and whiteflies. Aphids are the most significant virus vectors in temperate regions; whiteflies are very important in the tropics. Both aphids and whiteflies can be important greenhouse pests. Insects in other orders, including beetles and thrips, and noninsects such as eriophyid mites also are vectors of some plant viruses. The mechanism of virus transmission by these organisms is different from that of hemipterans, because their mouthparts and modes of feeding are different.

► **DID YOU KNOW?**

A single plant can be infected by more than one virus. The mode of insect transmission (nonpersistent or persistent) depends on specific interactions between a particular virus and insect vector. Hence, an insect vector may selectively transmit one virus but not another, even when both are present in the same plant.

Other agents can also create wounds and transmit viruses, including a few soilborne fungi and fungus-like organisms (*Olpidium, Polymyxa*, and *Spongospora*) and nematodes. The nematodes responsible for virus transmission are **ectoparasites** (see Chapter 4) that are migratory and move from plant to plant in the soil. As with hemipteran vectors, nematodes have stylets that they use to feed on root cells, and viruses are acquired and transmitted by the nematodes during feeding. People can transmit some viruses as they graft or prune plants or move machinery through a field.

Since the 1930s, various terms have been used to describe the transmission of plant viruses by insect vectors. Some terms refer to the relationship of the virus with the vector; others refer to the length of time that a vector can transmit a virus that it has acquired.

It is important to remember that these terms describe the mode of *transmission* of viruses, not the viruses themselves.

Two broad categories of insect transmission of plant viruses are **nonpersistent transmission** (also called **stylet-borne transmission**) and **persistent transmission** (also called **circulative transmission**). These terms were originally used to describe the transmission of viruses by aphids. The terminology works well for some other vectors and not so well for others. The two types of transmission are distinguished by the length of time it takes a vector to acquire and to transmit the virus and by the length of time the vector remains able to transmit the virus after it is acquired. In nonpersistent transmission, a virus can be acquired or transmitted by an insect in seconds, but the virus is usually retained by the insect for only a few hours. Many viruses that cause mosaic diseases are transmitted in this way. These viruses often infect cells near the surface of the leaf and therefore also can be transmitted mechanically (in sap). In persistent or circulative transmission, the insect must feed for minutes to hours to acquire or transmit the virus. The virus must "circulate" through the insect's body and eventually into the salivary glands (Figure 5.9), so there is a **latent period** between the time of acquisition and the time of potential transmission. However, the vector can transmit the virus for days, weeks, or even its entire life. Many viruses that cause yellows diseases are transmitted persistently. These viruses often multiply only in the phloem of their host plants and are therefore not mechanically transmissible. A few plant viruses are transmitted in a **persistent**, **propagative** manner, in which the virus also replicates in the cells of the insect vector.

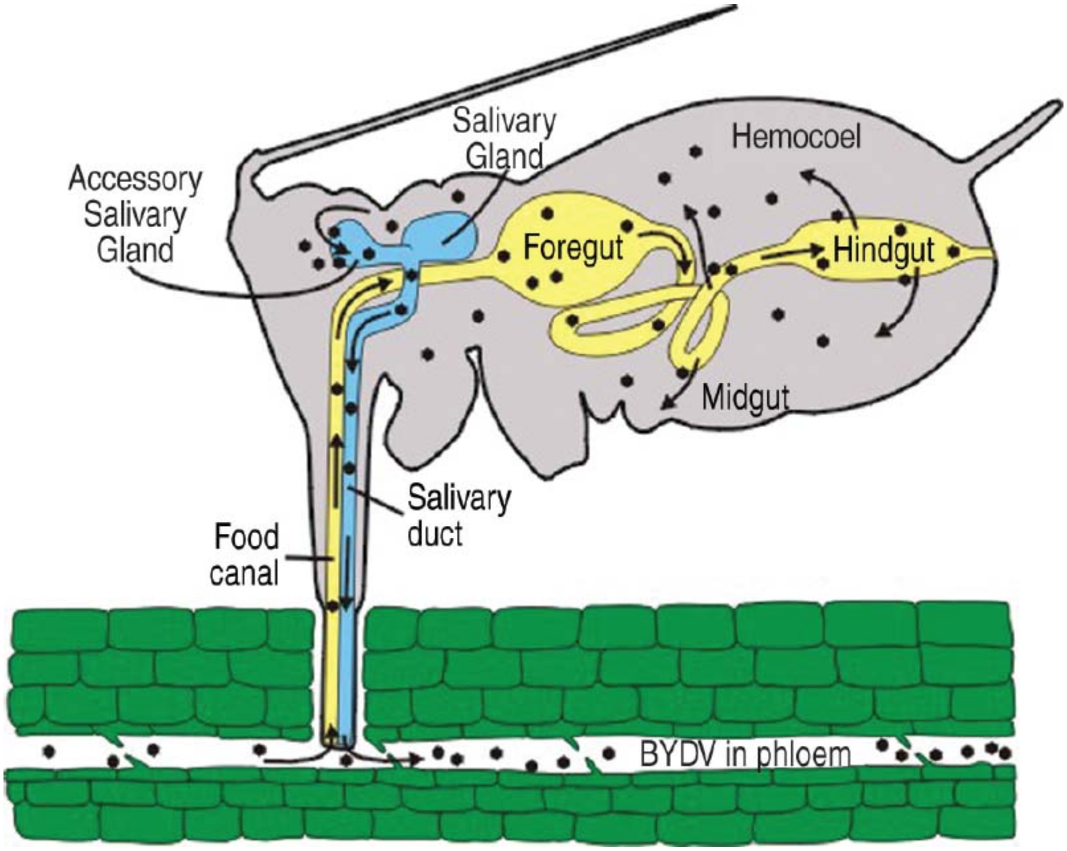
Salivary Gland
Hemocoel
Accessory Salivary Gland
Foregut
Hindgut
Midgut
Salivary duct
Food canal
BYDV in phloem

Figure 5.9. Aphid vector feeding on a plant host and the internal route of the viruses that cause barley yellow dwarf. These viruses are transmitted in a persistent (circulative) manner.

► DID YOU KNOW?

Some viruses, such as members of the *Rhabdoviridae* family, are able to multiply in both plants and insects. Plant virologists tend to regard insects as vectors carrying viruses from one plant host to another. From a different perspective, however, plants can be regarded as bridges that enable the movement of viruses from one insect to another. Some of these viruses cause no measurable harm to their insect hosts but cause distinct symptoms in plant hosts. This has led some scientists to conclude that these viruses have a long evolutionary history with insects and were introduced more recently into plants.

Types of Transmission of Plant Viruses by Insects

Type of transmission	Acquisition time	Retention time	Latent period?	Multiplication in vector?
nonpersistent (stylet-borne)	seconds to minutes	hours to days	no	no
persistent (circulative)	minutes to hours	days to months	yes	no
persistent (propagative)	minutes to hours	life of insect	yes	yes

Transmission of plant viruses to the next host generation is accomplished in several ways. Often, perennial plant hosts or overseasoning insect vectors serve as sources of inoculum in the next year. About 10% of plant viruses are known to be transmitted through **seed** of infected plants and a much lower number through **pollen** from infected plants. The percentage of infected seed produced on a plant varies with the time of infection, virus strain, host cultivar, and environmental conditions, but it is often very low. However, infected seeds provide a very early source of virus **inoculum** in a crop and can lead to significant losses if plant-to-plant spread occurs early in the growing season. Many viruses also are transmitted to the next generation through **vegetative propagation**. Crops such as potato, herbaceous ornamentals, small fruits and tree fruits, and most woody ornamentals are vegetatively propagated and are often infected by viruses. When parts of these plants (such as bulbs, corms, cuttings, rootstocks, or tubers) are used as planting stock, the next generation also is infected. The virus can even be moved around the world as plants and plant parts are shipped from place to place.

Transmission of Viruses to the Next Generation in

- debris of infected plants
- overwintering host plants, both crops and weeds
- seed
- overwintering vectors
- vegetative propagules (bulbs, corms, tubers)

Most plant viruses are transmitted from plant to plant by vectors, usually insects. Viruses are transmitted to a new generation through plant seeds or vegetative material or by remaining in overwintering hosts. Knowledge of the vector or vectors can help identify a virus.

How are virus diseases diagnosed?

Since the virus pathogen itself is invisible under a light microscope and can’t be grown on an artificial medium apart from its host, virus disease diagnosis is different from the diagnosis of fungal or bacterial diseases. As with diagnosis of diseases caused by other pathogens, however, information from the field or greenhouse where the problem occurs is often helpful. The species of plants affected and the pattern and timing of the spread of the disease often provide hints about the source and means of transmission of a virus. This information is useful in both diagnosis and disease management. As with other pathogens, proof that a particular virus is the cause of a disease is established by isolating (purifying) and transmitting the virus to healthy plants in which the disease is then reproduced (a modification of Koch's postulates, described in Chapter 1). Laboratory-based tests are also very important in diagnosis of plant virus diseases. These techniques include biological tests, electron microscopy, serological tests, and nucleic acid-based tests.

Koch's Postulates (Modified for Viruses)

1. Describe the symptoms and signs of a disease.
2. Purify the virus and note its characteristics.
3. Inoculate a healthy plant with the purified virus, and recreate the same disease originally described.
4. Purify the virus characterized in step 2 from the inoculated plant.

Biological tests, or **bioassays**, that use living plants are necessary to prove the pathogenicity of a newly discovered virus. The simplest bioassay uses an **indicator plant**, which is a host plant that shows an obvious reaction to virus infection. A **host range** test involves inoculating several different possible indicator plant species. Indicator plants that produce **local lesions** (Figure 5.10A) after inoculation give the fastest results, and each leaf can be used for a different assay (test). If a local lesion indicator plant cannot be found, it is necessary to wait for **systemic** symptoms (Figure 5.10B) to develop on the newest growth following inoculation. If the virus is sap-transmissible, as is TMV, plants are inoculated by rubbing either sap from a symptomatic plant or a purified virus preparation onto a leaf along with a mild abrasive. For a virus that is not sap-transmissible, such as *Barley yellow dwarf virus* (BYDV) (Figure

5.11), inoculation requires transmission by an appropriate vector or by grafting. Bioassays require propagation of plants and sometimes vectors, significant labor and space, and a minimum of several days for results, which may or may not be definitive. For these reasons, most routine virus disease diagnosis now relies on tests based on the unique structure and composition of a plant virus.

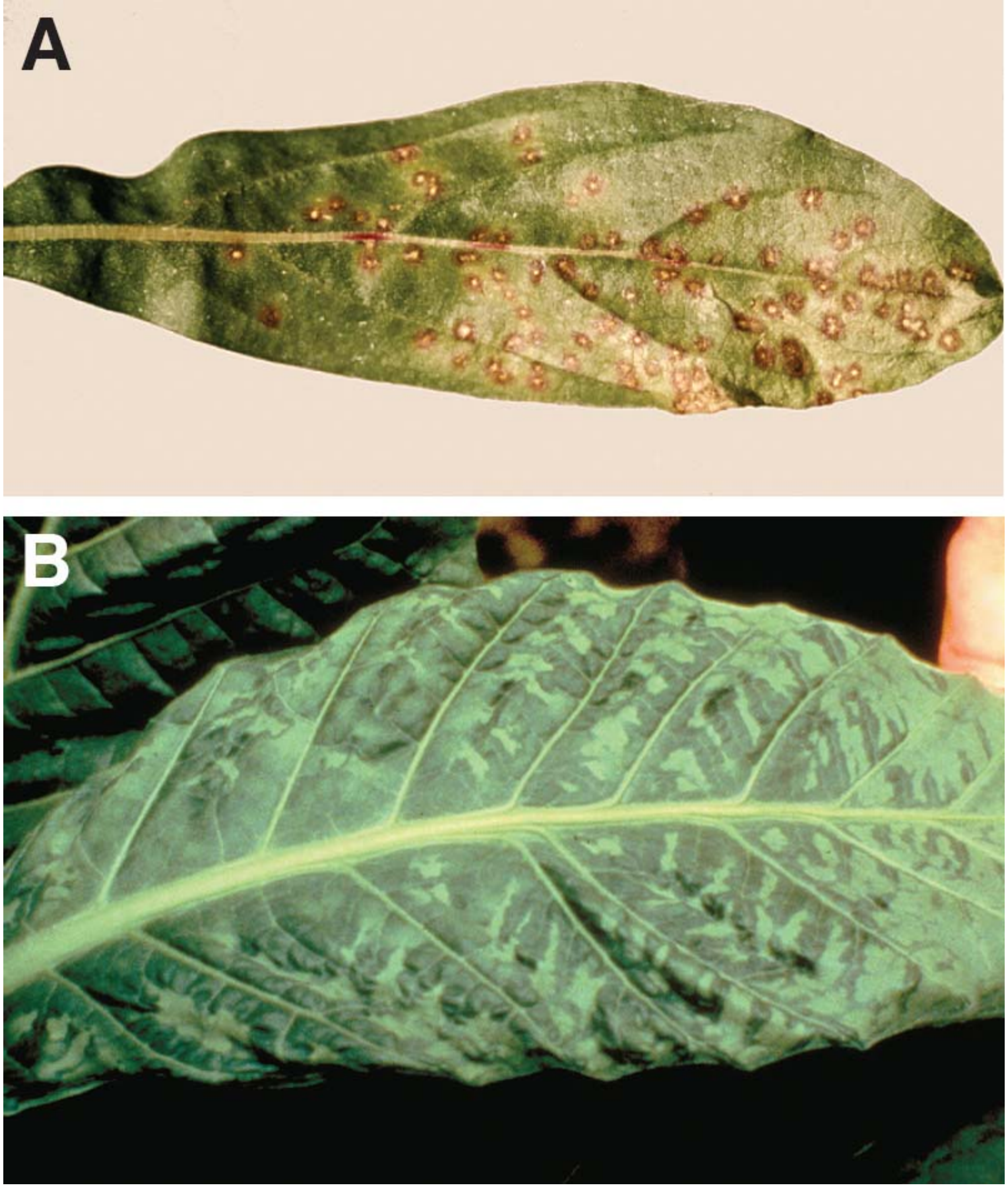

Figure 5.10. Local lesions (A) and systemic symptoms (B) resulting from virus inoculations.

► DID YOU KNOW?

In 1929, Francis O. Holmes (1899–1990) discovered that he could induce the formation of local lesions by *Tobacco mosaic virus* on hosts using a series of pins dipped in an inoculum of sap from infected plants. He found that the number of lesions produced was related to the concentration (or **titer**) of virus in the inoculum, thus developing a method to indirectly measure the amount of virus present. This first biological assay was used extensively to study many plant viruses and, later, to develop protocols for the purification of viruses.

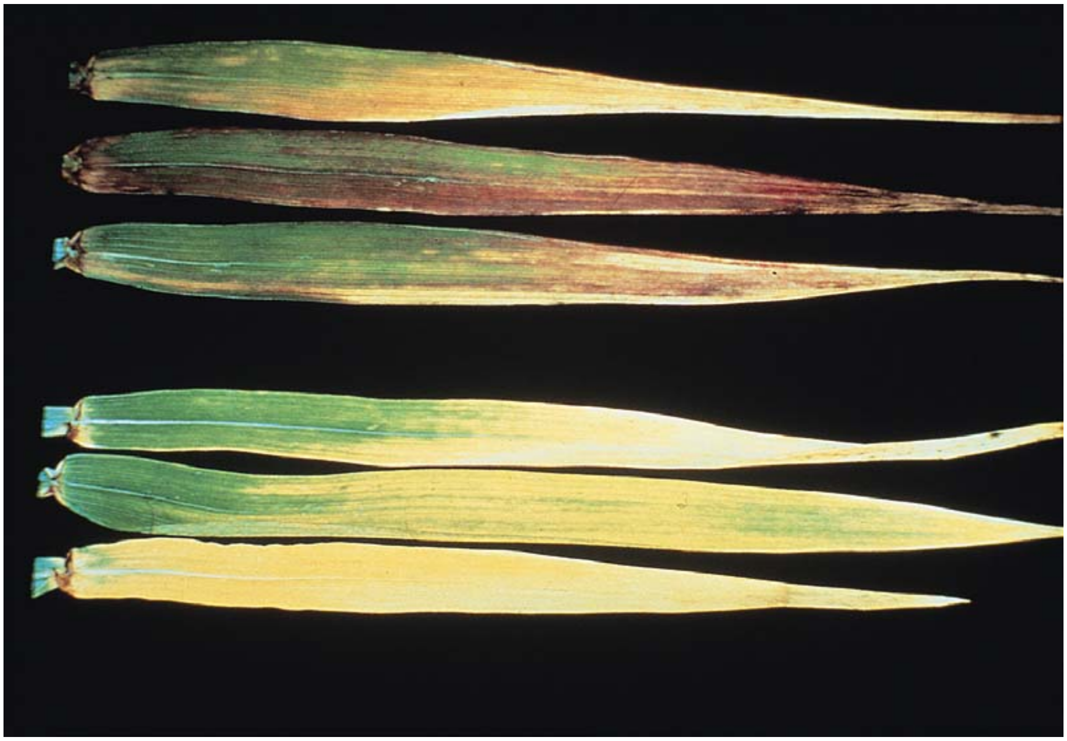

Figure 5.11. Leaf discoloration caused by *Barley yellow dwarf virus*.

DISEASE CLASSIC

Barley Yellow Dwarf

Hosts barley, oats, wheat, maize, rice, and other grasses

Pathogens *Barley yellow dwarf virus* (BYDV), *Cereal yellow dwarf virus* (CYDV)

Symptoms The most common symptoms are stunting and loss of green color from leaves, which appear yellow, orange, red, or purple. Older leaves are most severely affected. Less common symptoms include upright and stiff leaves, serrated leaf borders, reduced tillering and flowering, poor kernel filling, and sterility.

Disease Cycle BYDV and CYDV and their aphid vectors can overseason in winter cereals, volunteer crop plants, or weed hosts. The viruses can be introduced from local or distant sources by aphid vectors. Feeding by aphids creates the wounds required for virus infection. The virus particles replicate principally within plant cells in phloem tissue and move from cell to cell via plasmodesmata. The viruses are spread from plant to plant by persistent (circulative) transmission by aphids. There is no evidence of virus replication in the vector. Aphids feeding on phloem tissue acquire the viruses and retain them for days to weeks. Different strains of the viruses are transmitted most efficiently by different species of aphids.

Management The most widely used management strategy is planting resistant or tolerant host cultivars. Other strategies are effective in different parts of the world, including cultural practices (e.g., selection of a planting date that will allow a crop to avoid the disease) and biological and chemical management of aphid vectors.

Significance Barley yellow dwarf (also called oat red leaf) has been recognized as a disease since the late 1890s, but it wasn't until the 1950s that a viral etiology was proposed. This long delay in the recognition of BYD as a viral disease was due to differences between it and the classic mosaic diseases. BYD was one of the first and most widely studied of the yellowing diseases of plants, and it has been a model for detailed studies of the specificity of virus-vector interactions.

Plant Disease Profiles: Barley yellow dwarf

Inspection of a sample of sap from a suspected virus-infected leaf with the electron microscope, sometimes called a **leaf dip**, may reveal rod-shaped virus particles and some virus inclusions. These are unlike any normal plant cell component and, if present in high enough concentration, are easily identified among the cell contents. However, some spherical virus particles are similar in appearance to plant cell components, particularly ribosomes, and may be overlooked in a leaf dip. Despite this, a leaf dip is a quick and simple first test for virus presence in a diagnostic laboratory that has an electron microscope and an experienced microscopist.

► DID YOU KNOW?

An electron microscope uses a beam of electrons focused by electromagnetic lenses for visualization (instead of light focused by glass lenses). In an electron microscope, two points that are only 0.1 nanometer apart can be distinguished (resolved). This resolution is approximately 1,000 times more powerful than that of a light microscope. Electron microscopes have many uses in plant virology, including detection of virions in infected plants and plant extracts, elucidation of virus structure, study of the mechanisms of virus replication and pathogenesis, and examination of virus preparations to determine their purity.

Serological tests, or **immunoassays**, have been used for virus disease diagnosis for over 75 years. These tests rely on identification of a virus (the **antigen**) through its reaction with specific **antibodies**. Antibodies are molecules produced in animals by their immune systems as a defense against foreign macromolecules, including viral proteins and, to a lesser extent, nucleic acids. Virologists inject purified preparations of plant viruses into laboratory animals to induce the production of specific antibodies in the blood serum, which is then known as **antiserum**. After several weeks, the antibodies are harvested from blood samples taken from the animals. These antibodies, found in the antiserum, bind specifically to the plant virus used to create them and, to a lesser extent, to related plant viruses. A different antiserum must be produced for each virus. Some older serological tests rely on direct visualization of the antigen-antibody reaction, usually as the formation of a precipitate. These tests are rarely used today because a successful test requires high concentrations of virus and uses a relatively large amount of antiserum to produce a visible precipitate. More recently developed serological tests do not rely on the formation of a visible precipitate and therefore can detect lower concentrations of virus.

For many decades, all antisera were **polyclonal**, meaning that they contained a mixture of different antibodies that recognized different parts of the virus used to create them. Polyclonal antisera also contain all the other antibodies that were circulating in the animal's system when the sera were prepared, so the quality of the antiserum varies from one animal to another. More recently, virologists have used **monoclonal antibodies** (specific antibodies multiplied in tissue culture from a single original cell) that recognize only one part of a virus. In addition to this specificity, another advantage of monoclonal antibodies is that the cells in which they are produced are hybrids of an antibody-producing cell and a cancer cell that has indefinite life. These hybrid cells (**hybridomas**) can be frozen in liquid nitrogen, revived several years later, and used to produce another batch of identical antibodies. Rather than using the blood of lab animals as a source of antibodies, the antibodies can be mass-produced from cell cultures. Monoclonal antibodies are now used to quickly identify viruses and more complex pathogens such as bacteria and fungi (see Appendix 3 for details).

The most common serological test for the diagnosis of plant virus diseases is the **enzyme-linked immunosorbent assay** (**ELISA**). ELISA tests can use either polyclonal or monoclonal antibodies. There are many variations of ELISA, which is usually conducted in 96-well plastic microtiter plates (Figure 5.12). A common version of the test is **double-antibody sandwich ELISA** (**DAS-ELISA**), in which the wells are first coated with antibodies produced against viruses that the diagnostician believes may be responsible for the disease. Next, sap from the sample to be tested for virus is added to each well and incubated to allow time for any virions present to bind to the antibodies already attached to the well. The wells are washed after each step to remove unbound reagents. The **conjugate**, modified antibodies that can bind to sites on the virus, is added to each well. These antibodies are conjugated, or linked, to an enzyme, often alkaline phosphatase. If virions have become attached to the initial coating antibodies, the conjugated antibodies will bind to available sites on the particles, forming the “sandwich.” After the wells are washed again, an enzyme **substrate** is added, and a colored product develops when the enzyme portion of the antibody-enzyme conjugate acts on the colorless substrate. In the case of alkaline phosphatase, a yellow color that can be measured in a spectrophotometer develops in wells where virus is present. The intensity of the yellow color is related to the virus concentration.

DAS-ELISA Method

1. Coat the wells of a microtiter plate with the selected trapping antibody; incubate.
2. Wash the wells.
3. Add samples suspected of containing virus; incubate.
4. Wash the wells.
5. Add antibodies conjugated to an enzyme; incubate.
6. Wash the wells.
7. Add the enzyme substrate, incubate, and measure color intensity.

In Appendix 3, see diagram in Figure 2 and view the animation via this link.

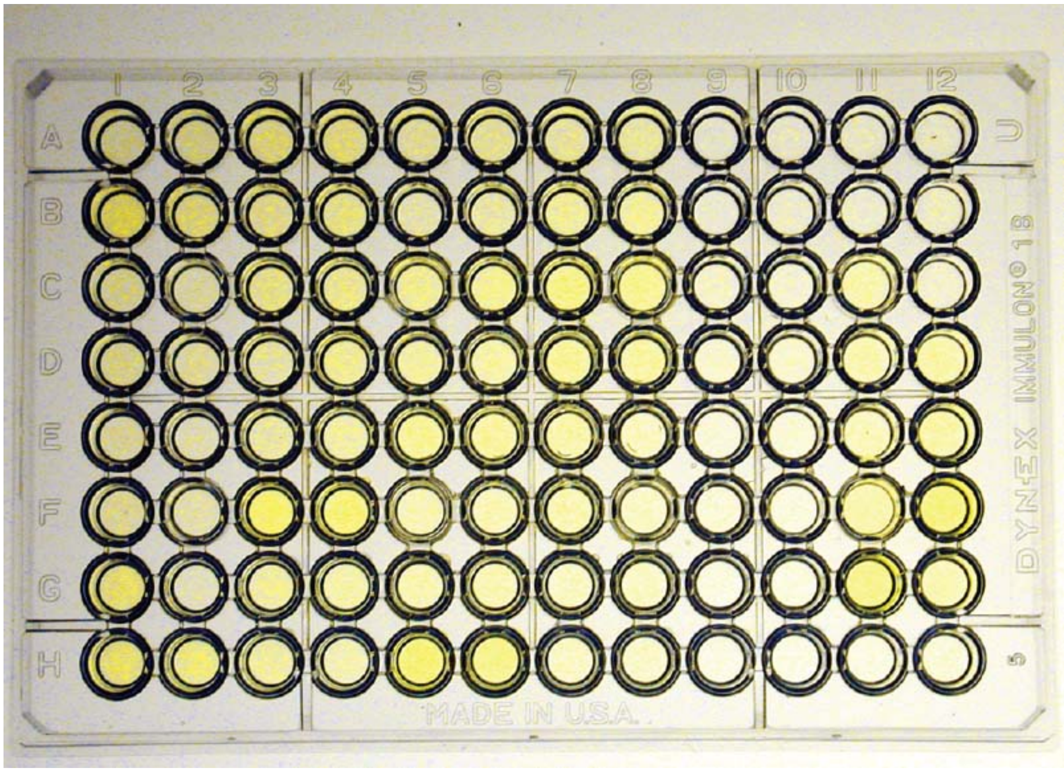

Figure 5.12. Microtiter plate used for enzyme-linked immunosorbent assay (ELISA). Wells containing virus develop color; wells without virus do not.

Advantages of ELISA include specificity, speed, and sensitivity due to the amplification of signal through the enzymesubstrate reaction.

Even very low virus concentrations can trigger the color reaction. This sensitivity is required for reliable detection of viruses that occur in low concentrations in plants, including many yellowing viruses such as BYDV. Many ELISA tests can be completed in a diagnostic laboratory in 1 or 2 days, depending on the availability of antisera. In addition, ELISA can be automated for large-scale testing, as is required in programs that certify that seed or vegetative plant parts are free of certain viruses.

Serological tests are based on the uniqueness of viral proteins. There also are diagnostic procedures based on detection of unique viral nucleic acids, including nucleic acid hybridization and **polymerase chain reaction** (**PCR**) (Figure 5.13). Since the 1980s, PCR has become widely used for plant virus disease diagnosis because of its relative simplicity and great sensitivity. As with ELISA, use of PCR in plant virus disease diagnosis relies on the diagnostician's knowledge of viruses that are likely to be the cause of the disease. In ELISA, this knowledge guides the selection of the antisera used in the assay; in PCR, it guides the selection of short pieces of DNA (containing 14–30 nucleotides) called **primers**, which can form base pairs or **anneal** with specific segments of the viral genome.

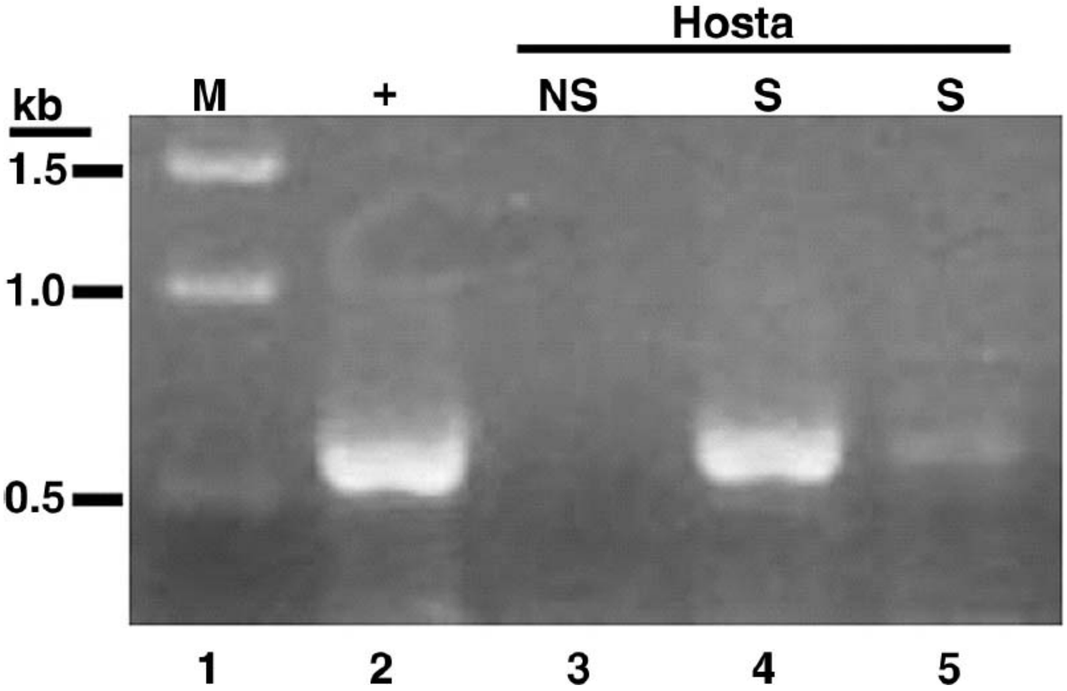

Figure 5.13. Detection of *Tomato spotted wilt virus* infection in hosta by reverse transcription polymerase chain reaction (RT-PCR).

For viruses with ssRNA genomes, the most common PCR assay is **RT-PCR** (reverse transcription PCR), in which an enzyme called a **reverse transcriptase** is used to make complementary DNA (cDNA) copies of the viral genome. Then, through a repeated cycle of annealing the primers to the cDNA to create short double-stranded DNA segments, extension of these segments by the enzyme *Taq* polymerase, and melting to separate the double strands, the concentration of selected portions of the viral nucleic acid can be multiplied more than a millionfold to produce readily detectable concentrations. Like ELISA, the specificity of PCR varies, depending on the uniqueness of the genome sections selected for amplification. Primers can be designed to detect a single virus or a whole family of viruses. Like ELISA, PCR is also used to detect other kinds of pathogens (see Appendix 3). **Viroids**, which are small, infectious, single-stranded, circular RNAs (see Viroids box), cannot be detected by serological techniques, because they lack proteins; diagnosticians rely on nucleic acid-based methods such as PCR for this group of pathogens.

Viroids

- small, infectious, single-stranded, circular RNAs
- encode no proteins and therefore have no coat (viroids in seeds and plants cannot be detected by immunoassays)
- symptoms similar to those caused by viruses, including mottle, stunting, and deformation
- transmission by contaminated tools and equipment (mechanical transmission), by vegetative propagation, and in seeds and pollen

Example diseases: cadang-cadang of coconut, chrysanthemum stunt and chlorotic mottle, citrus exocortis, potato spindle tuber, scar skin of apple, sunblotch of avocado

RT-PCR Method

1. Use reverse transcriptase to make DNA complementary to the viral RNA genome (cDNA).
2. Anneal selected primers to the cDNA, creating short, double-stranded sections.
3. Extend the double strands using *Taq* polymerase.
4. Melt the double strands into single strands by raising the temperature.
5. Repeat steps 2 through 5 many (e.g., 25–35) times.
6. Detect the amplified fragments of DNA on a gel made of agarose or polyacrylamide.

In Appendix 3, see diagram in Figure 5 and review the animation via this link.

The method chosen to detect a plant virus often depends on the goals of the program or the study. If the goal of a certification program is to select only plants that are virus-free, then sensitivity is the prime criterion, and a test like PCR may be used. If a very large number of samples must be processed, for example, in a large epidemiological study, then rapidity and cost-effectiveness are key considerations, and ELISA may be the method selected. Diagnostic labs may stock primers and antisera of common and important viruses and send plant material to specialty labs for more complete diagnostic testing as needed. Leaf dips and bioassays are still sometimes used as the first steps to determine whether a disease is caused by a virus. These methods also are useful for detection of viruses for which antisera and primers are not yet available. For these reasons, no single method is universally used for plant virus disease diagnosis.

The most common techniques used for diagnosis of virus diseases rely on detection of either the viral protein (by serological assays, such as ELISA) or the viral genome (by nucleic acid assays, such as RT-PCR). A leaf dip viewed in an electron microscope can provide information that may lead to a quick diagnosis; however, more time-consuming biological tests (bioassays) remain the only means to prove that one specific virus is the cause of a particular disease.

► DID YOU KNOW?

ELISA and PCR are widely used in human medicine, as well as in plant pathology. Variants of these assays are used to diagnose human diseases (including AIDS, herpes, and Lyme disease), to detect deleterious genetic mutations, and to identify specific drugs, metabolites, and hormones in body fluids (for example, in home pregnancy test kits).

Virus Assays

Assay type	Time required	Advantages	Disadvantages
bioassay	days to months	sensitive, proof of pathogenicity	plants and in some cases vectors-required; often does not identify the specific virus present
ELISA	hours to days	sensitive, specific, safe	specific antibodies required
PCR	minutes to hours	sensitive, specific	specific primers required

How are virus diseases managed?

Once a plant is infected with a virus, it is usually not possible to cure it. Some individual plants or parts of plants have been cured through the use of heat or chemical treatments, but there are no “viricides” available for commercial use in a garden, greenhouse, or field. The difficulty in curing a plant of a virus lies in the intimate interactions between the virus and its host. Since the virus uses host cell metabolic pathways for genome replication and protein synthesis, blocking these pathways will cause harm to the host as well as to the virus. Therefore, most plant virus disease management focuses on prevention.

Strategies used for plant virus disease management may focus on the host, the virus, the environment, or the vector, if one is involved. Usually, multiple methods are required for effective management.

Avoidance

It is sometimes possible to avoid viruses or to ameliorate their effects by planting a susceptible crop at a different time or in a different location than usual. This management strategy is particularly useful for diseases spread by seasonal vectors. The persistence of vectors in the tropics and subtropics complicates virus management. If crops are planted earlier, seasonal vectors arrive when the plants are older and less susceptible. If crops are planted at a distance from where vectors overseason, the vectors may arrive late or not at all.

Exclusion

Some virus diseases can be managed by keeping them out of an area or a crop for a period of time. **Long-term exclusion** often involves federal and state quarantine laws that forbid the movement of infected plants or plant parts into an area. This strategy is particularly useful in areas that are geographically distant from virus sources and for viruses that are transmitted via seed and vegetative parts. Detection of virus infection requires laboratory tests, so effective quarantines must be supported by extensive facilities and personnel and by reliable and sensitive tests for virus detection. Quarantines may be effective for years or decades but, given our highly interconnected and mobile global community, they are likely to fail eventually. The recent introduction of *Plum pox virus* (PPV) into North America from Europe is one example. Thus, quarantines are best viewed as temporary measures that afford virologists time to explore other management strategies.

Short-term exclusion is usually based on planting seed or stock certified to be virus free. These planting materials are produced through extensive testing and therefore cost growers more. If the higher yields obtained from certified seed or planting stock more than compensate for the higher initial cost, this strategy is worthwhile. **Certification** schemes have been developed for many vegetatively propagated crops, such as potato. One success story is the reduction of lettuce mosaic, caused by *Lettuce mosaic virus* (LMV), in California, where yields increased from 140 to 190 cartons per hectare after the use of virus-free seed was introduced.

► DID YOU KNOW?

The potatoes that you buy at the store are the product of intensive disease management strategies. Potatoes are vegetatively propagated from small tubers or sections of tubers called "seed pieces." Once virologists realized the adverse effect of viruses on potato yields, they used heat treatment and meristem tip culture to produce "nuclear stocks" of virus-free potatoes. Nuclear stocks are maintained in carefully controlled environments and are increased in fields where the virus vectors, often aphids, arrive late in the growing season. Any virus-infected potatoes are eliminated through careful visual inspections and extensive virus testing procedures. The resulting "certified" potatoes are planted in production fields for potatoes sold to processors and to the public in grocery stores. With all the work that goes into their production, potatoes are a very inexpensive source of good nutrition!

Eradication

Some plant virus diseases are managed by destroying their host or vector. **Eradication** can be done by chemical or physical means. Herbicides can be used to kill unwanted hosts of a virus or vector, such as weeds or volunteer crop plants. This strategy is impractical for the management of viruses that have a very broad host range, such as BYDV, which infects over 200 grasses. Remains of plants infected with mechanically transmissible viruses such as TMV should be destroyed. Insecticides can be used to kill overwintering viruliferous vectors. The effectiveness of this management strategy depends on killing the insects before they have the opportunity to transmit the virus to crop plants, so it is more often effective with persistently transmitted viruses.

Virus-infected plants also can be physically eliminated. **Roguing**, the removal of infected plants from a population, has been most successful in tree crops in which a virus disease has a slow rate of natural spread and both disease detection and tree removal are rapid. Heat treatment and **meristem tip culture** (Figure 5.14) are methods used to produce virus-free individual plants from virus-infected plants. Hot-water treatment has been used to eliminate viruses from dormant plant parts, such as tubers or budwood; hot air is more commonly used to eliminate viruses from actively growing plants. Meristem tip culture involves the excision of 0.1–0.5 millimeter of the growing tips of the virus-infected plants. Some meristem tips are virus free; others can be freed from virus by heat treatment, which is believed to reduce virus replication. The virus-free meristem tip must then be regenerated into a virus-free plant, which can be propagated in environments that exclude vectors and other means of virus infection (Figure 5.15). These techniques have been used to create virus-free planting material of many crops, including apple, peach, potato, strawberry, many herbaceous and woody ornamentals, and greenhouse flower crops, such as chrysanthemum.

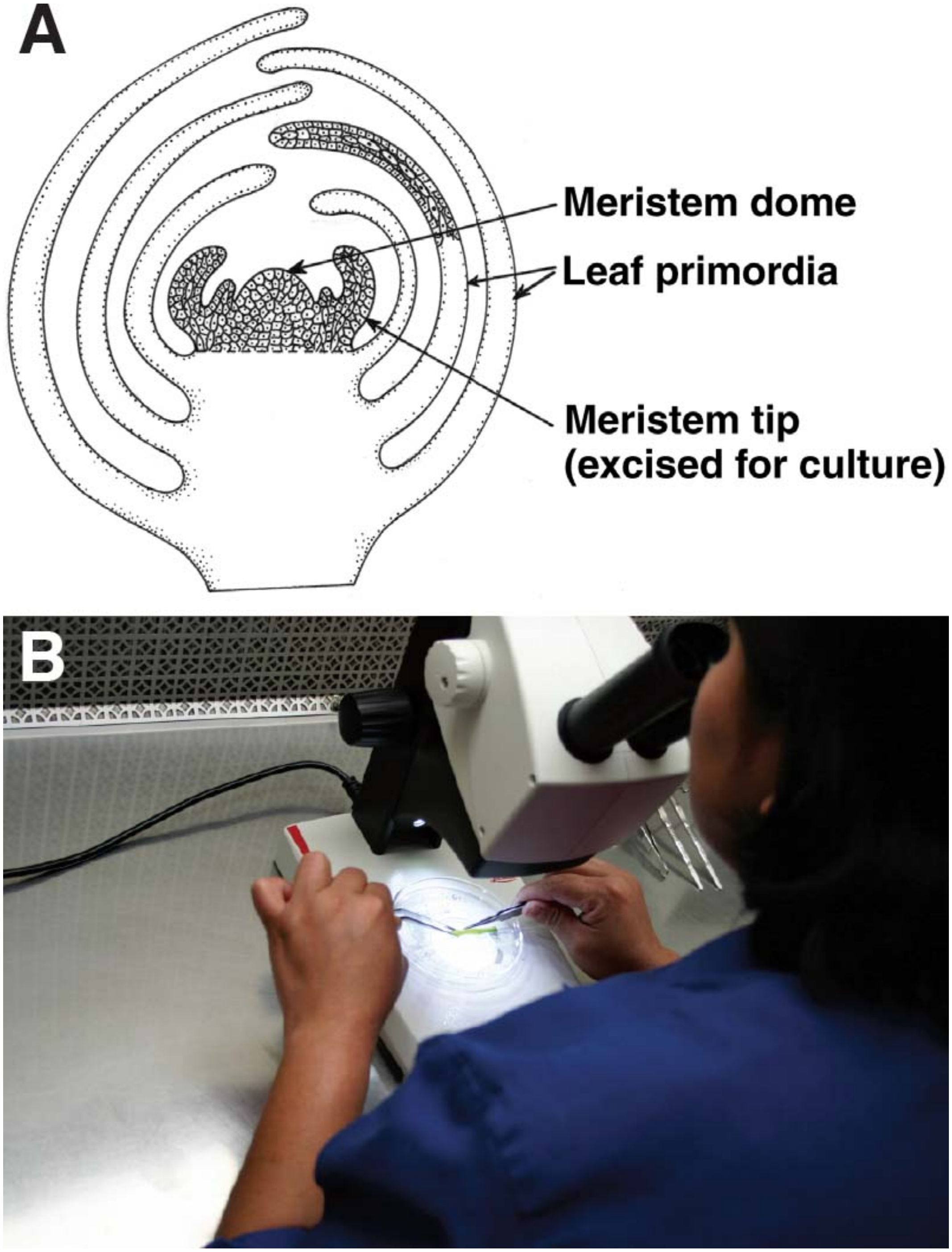
A
Meristem dome
Leaf primordia
Meristem tip
(excised for culture)
B

Figure 5.14. Meristem tip culture. A, Cross section of a bud, showing the meristem tip region (above the dotted line). B, Removal of a meristem tip

with the use of a dissecting microscope.

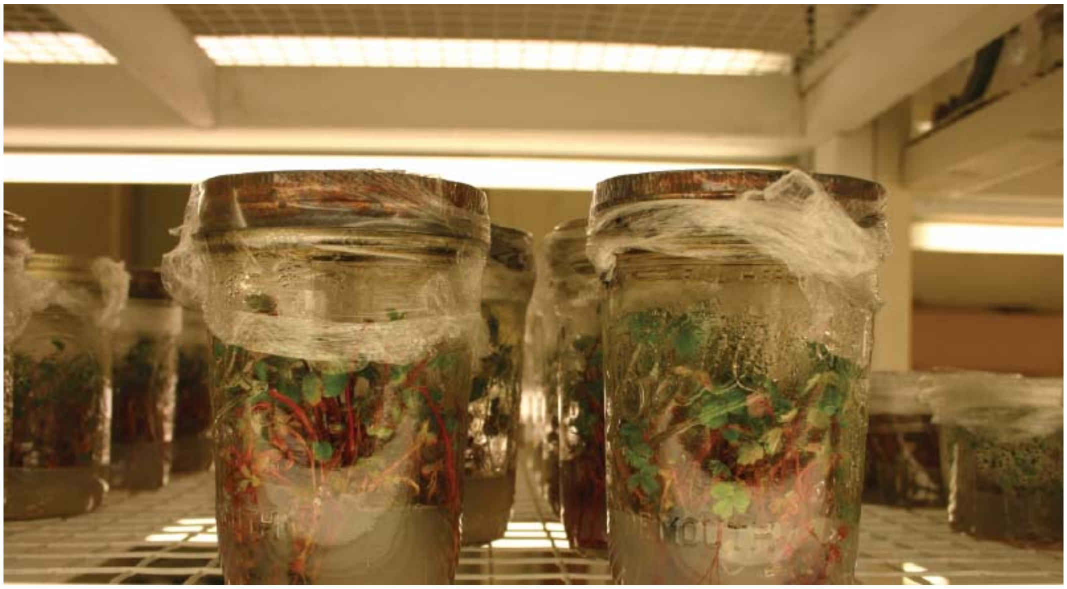

Figure 5.15. Tissue culture jars in a laboratory where thousands of plants are propagated in a virus-free environment.

Protection

Although there are no chemicals that act directly against plant viruses, **protection** can be successfully implemented in other ways. Cultural strategies designed to protect crops from vectors include mandated crop-free periods when all growers in an area leave their fields fallow, the use of barrier crops planted around field edges, and placement of reflective mulches (e.g., silver polyethylene) in crop rows. Fallow periods reduce vector populations in the area; barriers and mulches reduce the number of insects reaching the crop. The effectiveness of these strategies depends on many factors, including the host range of the virus, the type of transmission involved, the relative cost and benefit of the strategy to growers, and growers' coordination and willingness to adopt new agronomic practices.

Insecticides can be used to protect plants from insect vectors that spread virus diseases. This management strategy is most successful for persistently transmitted viruses, because vectors are killed before they have time to acquire and transmit the virus. Horticultural oil is one chemical that can protect plants against infection by some viruses that are nonpersistently transmitted by aphids. To be effective, plant tissue must be resprayed with the oil after storms and during growth periods. The relatively high cost of this management strategy limits its use to high value crops, such as flowers, fruits, and vegetables.

► **DID YOU KNOW?**

Only some diseases caused by insect-transmitted viruses can be effectively controlled by the application of insecticides. For example, some attempts to eradicate aphid vectors of *Potato virus Y* (PVY) with insecticides not only failed to control the disease but actually *increased* its spread! Scientists found that when insecticide was present, the aphids became restless and moved more often from plant to plant, making brief probes with their stylets, rather than settling down to feed on a single plant. The increased vector movement and probing increased the nonpersistent transmission of PVY.

Cross-protection is a biological phenomenon that was first described in the 1920s, when several virologists reported that a plant infected with a mild strain of a virus could not be infected with a more severe strain. Cross-protection has been used commercially to protect greenhouse-grown tomato plants from infection by TMV and orchard-grown citrus from *Citrus tristeza virus* (CTV). More importantly, cross-protection has provided clues to how plants can be genetically modified to resist plant virus infection.

Resistance is a very important strategy for the management of diseases caused by viruses, as it is for diseases caused by all other plant pathogens. Plants with genes for resistance to a particular virus or to its vector may be selected from plant populations, or resistance genes from related plants may be incorporated through breeding programs. When looking for virus-resistant plants, sometimes plants will be found that have **immunity** to a specific virus; these plants do not allow any virus replication, even in single cells. In contrast, resistant plants may limit the spread of a virus, resulting in localized infections, often seen as local lesions. Some host plants may show no obvious symptoms of virus infection, although the virus is able to multiply and spread within them. This **tolerance** is also under genetic control. Tolerance is a less favorable means of virus disease management than resistance, because the virus is still present in the crop and available for spread to other susceptible crops or interactions with other viruses, which can result in severe disease due to **synergism**.

Host Plant Reactions to Viruses

- A plant is **immune** to infection by a virus if the virus does not multiply in it, even in individual isolated cells called protoplasts.
- A **resistant** host restricts virus multiplication or spread.
- A **tolerant** host produces a good yield in spite of unrestricted virus infection.
- A **susceptible** host is unable to restrict virus multiplication or spread and shows obvious symptoms, including yield loss.

Resistance to each of the important virus pathogens of a plant is desirable. However, the selection of virus-resistant plants in traditional breeding programs can take many years, and virus resistance genes are not always available. Virologists can now identify and isolate genes that confer resistance to virus diseases and introduce them into susceptible plants, using laboratory techniques. This seems to work quite well with plants in the same species and less well or not at all with plants in other species, genera, and families. To more quickly obtain virus resistance, virologists have developed **transgenic plants** with resistance genes from naturally resistant plants, viral sequences, and other sources that block processes required for virus replication. One of the earliest and most commonly used strategies was to introduce the gene for the viral coat protein into the host plant (Figure 5.16). This strategy was first used successfully for TMV and has been used commercially to manage other viruses in crops such as papaya, potato, and squash. Other strategies to create genetically modified virus-resistant plants include the use of other viral nucleic acids and genes for antibodies to plant viruses. All of these strategies are strictly regulated, and field tests are carefully monitored to ensure that the benefits of this new technology outweigh the risks (see Chapter 9 for more details).

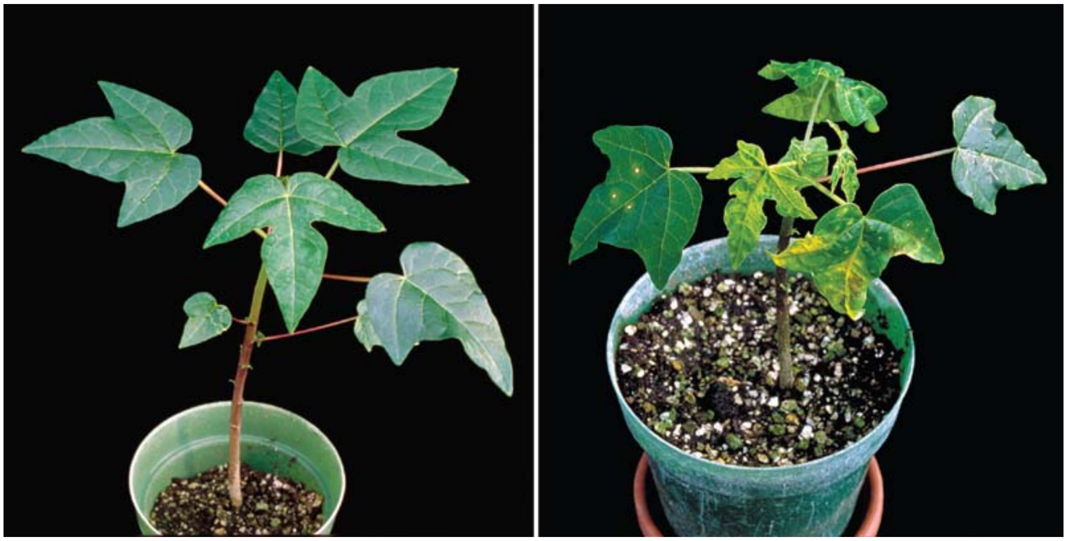

Figure 5.16. Transgenic papaya (left) and nontransgenic papaya (right) inoculated with *Papaya ringspot virus*. The transgenic papaya contains the gene for the viral coat protein, which confers protection from the virus.

As with all plant diseases, virus diseases are often most effectively managed through a combination of strategies, or an **integrated pest management** program. The most effective implementation of these management strategies relies upon accurate disease diagnosis and forecasting (see Chapter 11).

► DID YOU KNOW?

Infection of wheat by *Wheat streak mosaic virus*, a member of the *Potyviridae* family, caused significant yield losses in Alberta, Canada, where wheat was grown year-round. Spring wheat growing in the fields from April until August and winter wheat growing from August until June or July provided continuous hosts for the virus and its tiny mite vectors. Wheat growers in Alberta agreed to leave fields fallow for the last two weeks of August in hopes that the mites would die in the absence of plant hosts. The growers also eradicated volunteer wheat and barley plants and grass hosts of the mites. Their cooperative strategy successfully prevented infection of the fall-planted crop and has disrupted the disease cycle of this virus.

Management Strategies for Virus Diseases

Avoidance

- Plant at a location where the virus or vector is not present.
- Time planting so that plants are at a less susceptible growth stage when the virus or vector is present.

Exclusion

- Impose quarantines (local and international).
- Use virus-free seed or stock.

Eradication

- Use herbicides to kill weed hosts.
- Rogue infected plants.
- Use insecticides to kill vectors.
- Use heat treatment and/or meristem culture to produce virus-free plants.

Protection

- Mandate crop-free periods.
- Plant barrier crops.
- Use reflective mulches.
- Apply insecticide or oil.
- Inoculate plants with a mild virus strain to provide cross-protection.
- Plant resistant or tolerant hosts.

Successful management of plant virus diseases is achieved by several strategies, including avoidance, exclusion, eradication, and protection. Often, virus diseases are most effectively managed through a combination of strategies, and the most effective strategy is to *prevent* plants from becoming infected with viruses in the first place.

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Questions

1. What are the main characteristics of a plant virus? How do viruses differ from fungi, bacteria, and nematodes?
2. Why are all viruses considered obligate parasites? What other types of plant pathogens are obligate parasites?
3. Give an example of a symptom caused by viruses that is distinct from those caused by other types of pathogens. Give an example of a symptom caused by viruses that is similar to those caused by some other types of plant pathogens. Are symptoms useful for the diagnosis of virus diseases of plants?
4. You receive a plant that is yellowed and stunted. These symptoms can be caused by a virus, a phytoplasma, or a nutrient deficiency, among other causes. What methods can you use to determine which of these is the cause of the problem?
5. If you were a plant virus, how would you prefer to be transmitted from plant to plant? From generation to

generation? Why? Remember, the goal of any biological entity is to survive and multiply!

6. How long does it take for a virus to be transmitted by an aphid? Explain your answer. Why are insecticides not recommended for preventing the transmission of many plant viruses by insect vectors? What are three additional ways in which viruses are transmitted from plant to plant?
7. ELISA and PCR are sensitive methods used for virus detection and disease diagnosis. What are some advantages and disadvantages of each method? Describe a situation in which you would choose to use ELISA and one in which you would select PCR. When would you use a bioassay instead of either of these methods?
8. Why is management of plant virus diseases largely based on prevention rather than cures?

Challenge Questions

1. You have read about fungi, bacteria, phytoplasmas, nematodes, and viruses as plant pathogens. In the list below, which group of organisms in the second column is most closely related to the organisms in the first column? Explain why.

bacteria:	fungi or viruses
fungi:	nematodes or bacteria
phytoplasmas:	bacteria or viruses

2. For decades, diseases caused by phytoplasmas were thought to be caused by viruses. Explain what caused this confusion.
3. In spite of the fact that viruses rarely kill their host plants, virus testing of plants using DNA tests, immunoassays, and bioassays is widely practiced. Why is accurate detection of viruses in plants so important?

4. You receive a package that contains a plant that a grower suspects is either infected with a virus or has herbicide injury. What additional information would be helpful and what lab procedures might you use to solve this problem without sending the sample elsewhere?
5. Pruning is often recommended for bacterial and fungal infections of woody plants, but not for virus-infected plants. Why is this practice not recommended for virus disease management?
6. Most virus management involves preventive measures rather than curative ones. Explain why it is difficult to cure plants of virus infections.

Supplementary Resources

Illustrated Glossary

Special Topic

- Introduction to plant viruses, the invisible foe
  - Introducción a los virus vegetales, el enemigo invisible (Spanish)

Plant Disease Profiles

- Barley yellow dwarf
- Cucumber mosaic
- Potato spindle tuber
- Soil-borne wheat mosaic
- Tobacco mosaic
- Tomato spotted wilt

Laboratory Exercise

Antigen-antibody testing: A visual simulation or virtual reality
Tobacco mosaic virus

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

antibody
antigen
antiserum (pl. antisera)
avoidance
bioassay
breaking
capsid
certification
chlorosis
circulative transmission
coat protein
cross-protection
cytopathology
deoxyribonucleic acid (DNA)
dwarfing
ectoparasite
electron microscope
enation
encapsidate
enzyme-linked immuno-sorbent assay (ELISA)
eradication
exclusion
flexuous
flower break

host range
hybridoma
icosahedral
immune
immunoassay
inclusion body
indicator plant
inoculum
integrated pest management
latent period
leaf dip
leafroll
local lesions
mechanical transmission
meristem
meristem culture
messenger RNA (mRNA)
monoclonal antibody
mosaic
mottle
movement protein
multiparticulate
multipartite
necrosis
negative-sense (–sense) RNA
nonpersistent transmission
open reading frame (ORF)
persistent transmission
phloem necrosis
plasmodesma (pl. plasmodesmata)
polyclonal antibody
polymerase chain reaction (PCR)
polyprotein

positive-sense (+sense) RNA
post-transcriptional gene silencing (PTGS)
primer
propagative transmission
protection
replicase
replication
resistant
reverse transcriptase
ribonucleic acid (RNA)
ringspot
rogue
rugose
sap transmission
serology
stem pitting
strain
stunting
stylet
stylet-borne transmission
subgenomic RNA
substrate
synergism
systemic
titer
tolerance
transfer RNA (tRNA)
transgenic
translation
tumor
vector
vegetative propagation
vein banding

virion
viroid
virus
wound
yellows

Exercises

Exercise 5.1. Identify each of the common virus symptoms shown in the photographs.

Exercise 5.2. Identify each of the common virus vectors shown in the photographs.

Internet Resources

APS Resources

The *Bemisia tabaci* complex: Genetic and phenotypic variability drives begomovirus spread and virus diversification
Plum pox potyvirus disease of stone fruits
Tobacco mosaic virus: The beginning of virology
Transgenic virus resistant papaya: New hope for controlling *Papaya ringspot virus* in Hawaii
Viroids: New and continuing risks for agricultural crops

Recommended Websites

All the Virology on the WWW
The Big Picture Book of Viruses
PCR Animation
Plant Viruses Online

Internet Research

Internet Research Exercise 5.1. From the APS *Common Names of Plant Diseases*, select a virus disease of an annual herbaceous plant. Consult Plant Viruses Online, the Internet, and other resources to find answers to the following questions:

a. What is the name of the virus?
b. What host plants does the virus infect?
c. What shape is the virus, and of what kind of nucleic acid is the genome composed?
d. How is the virus disseminated horizontally (from plant to plant) and vertically (from one generation to the next)?
e. If there is an insect vector, what is the means of transmission (nonpersistent or persistent)?
f. What management practices are recommended?

Internet Research Exercise 5.2. Choose a vegetatively propagated crop, such as strawberry or another small fruit, apple or another tree fruit, a woody ornamental, potato, or a greenhouse flower crop, such as poinsettia, chrysanthemum, carnation, or geranium. List the three virus diseases that are most destructive to this crop. How is each of these virus diseases managed in this crop?

Internet Research Exercise 5.3. Agdia is one of several companies that specialize in pathogen detection tests. Go to the Agdia website and click on “Testing Services” and then “Crop Screens.” Determine how many virus tests are available for each of three crops. What detection methods are used?

CHAPTER 6

What Are the Causes of Plant Diseases?

Parasitic Flowering Plants

IN THIS CHAPTER:

What are parasitic flowering plants?

What symptoms and signs do parasitic flowering plants cause?

How do parasitic flowering plants survive and spread?

How are diseases caused by parasitic flowering plants diagnosed?

How are parasitic flowering plants managed?

What are parasitic flowering plants?

Certain flowering plants (angiosperms) are parasites of other plants, but in all other respects they have the typical life cycle of other angiosperms. Seeds germinate and develop into plants with flowers that are pollinated and eventually produce more seeds. One can think of these plants as extreme weeds, obtaining water, minerals, and sometimes photosynthates directly from their host plants. Parasitic flowering plants produce specialized absorption structures called haustoria. (This same term is used for the nutrient-absorbing structures produced by some fungi.)

Some parasitic flowering plants (hemiparasites) have green leaves and produce their own food through photosynthesis. They absorb water and minerals via haustoria from their hosts and are primarily xylem parasites.

Other parasitic flowering plants (holoparasites) are totally dependent on their hosts and do not produce food by photosynthesis (Figure 6.1). The haustoria of these biotrophs (obligate parasites) invade both the xylem and phloem of their hosts to absorb everything the parasite requires for life. Holoparasites typically have very small leaves called scales and are not green, but they still produce flowers and seeds. There are, of course, exceptions, and some parasitic flowering plants do not fit exactly into either of the two categories as hemiparasites or holoparasites.

Parasitic Flowering Plants

hemiparasites

- parasites of xylem
- use water and minerals from host
- usually green and leafy
- capable of photosynthesis

holoparasites

- parasites of xylem and phloem
- totally dependent on host
- not green, with small leaves called scales
- not capable of photosynthesis

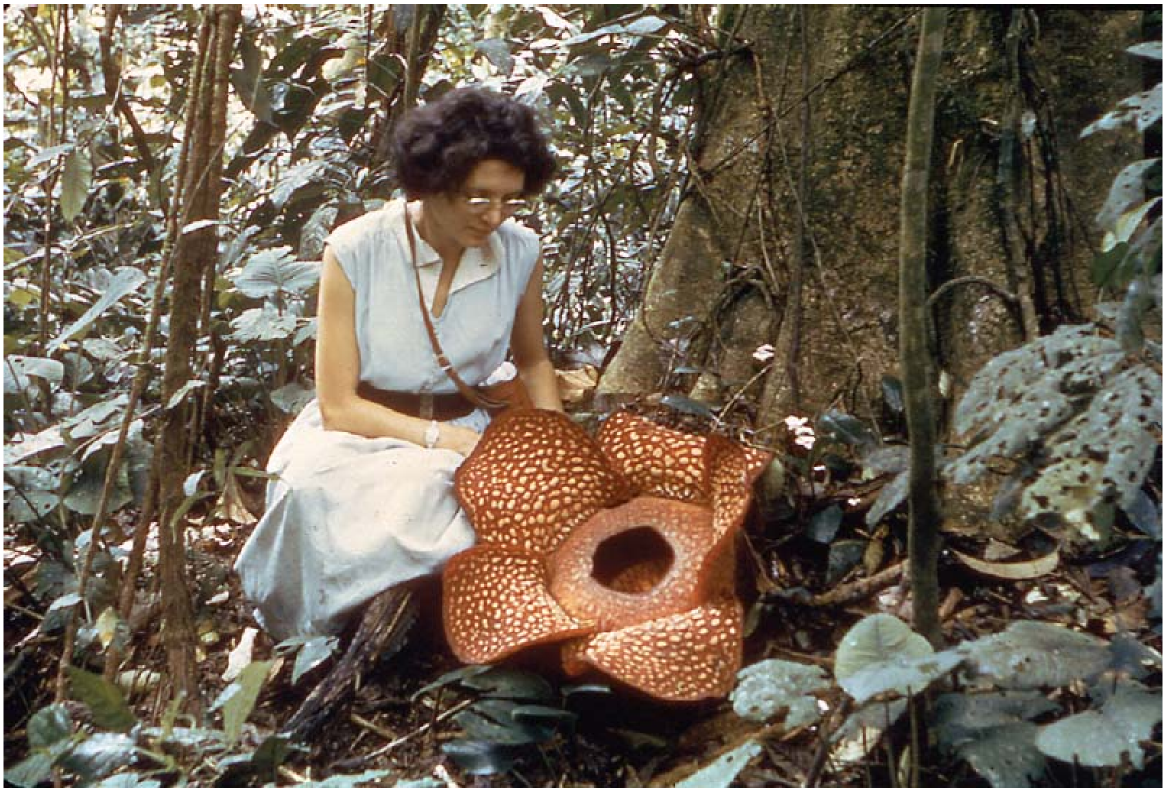

Figure 6.1. Rafflesia arnoldii, a holoparasitic plant.

DID YOU KNOW?

The biggest flower in the world is produced by Rafflesia arnoldii (Figure 6.1), a parasitic plant found only in the rainforests of Indonesia. It is a holoparasite of a tropical vine and obtains food and water from a host plant to produce flowers that are up to a meter (about 3 feet) across. The flowers give off a fetid odor, which attracts pollinating flies. Because of its terrible smell, it is sometimes called the corpse plant. R. arnoldii is dioecious; that is, it produces separate plants with either male flowers or female flowers. Both kinds of flowers are malodorous.

In ancient times, people recognized that parasitic plants were unusual and sometimes assigned special powers to them. One of the most well known is the Christmas mistletoe (Figure 6.2). Mistletoe appeared magical, perhaps because it was sometimes found in oak trees, which themselves were considered mystical. In addition, leafy mistletoes remain green during the winter and never grow in the soil, as other plants do. Mistletoe was an important part of the summer solstice rituals of the Druids and had roles in the mythology of the Celts, Greeks, Norse, and Romans. Mistletoe was considered to have medicinal value as a universal healer. Beliefs about its mystical properties led to the more recent custom of kissing under the mistletoe at Christmas (see the APS feature listed under Internet Resources).

Figure 6.2. Leaves, stems, and berries of mistletoe (Phoradendron serotinum).

Parasitic flowering plants are biotrophs (obligate parasites). They have modified roots with haustoria for absorption. Hemiparasites are green and photosynthesize, but they absorb water and minerals from the xylem of their hosts. Holoparasites are not photosynthetic and absorb water, minerals, and photosynthates from the xylem and phloem of their hosts.

What symptoms and signs do parasitic flowering plants cause?

Although there are many known parasitic flowering plants, only a few are economically significant because of their impact on host plants. Symptoms on affected plants are similar or identical to those of plants growing in intense competition with weeds: wilting, stunting, and nutrient deficiency symptoms. In some cases, the normal growth pattern of the host is affected. A proliferation of branches from a central point, called a witches’ broom, is a common symptom on conifers infected by dwarf mistletoes (Figure 6.3).

Figure 6.3. Ponderosa pine (Pinus ponderosa var. scopulorum) with a witches’ broom caused by dwarf mistletoe (Arceuthobium vaginatum ssp. cryptopodum).

DID YOU KNOW?

Some witches’ brooms of conifers are caused by plant mutations rather than dwarf mistletoe infections. Mutation-induced witches’ brooms are sometimes used to propagate dwarf conifers for landscapes. Witches’ brooms in plants also can be caused by fungi, phytoplasmas, and certain arthropods.

A parasitic flowering plant itself is the sign of the pathogen. Some parasitic flowering plants attach to aboveground parts of their hosts, where they are easily noticed. Other species are root parasites. These parasitic flowering plants usually emerge from the soil near the base of a host plant, but their belowground connection to the roots of the host plant may not be obvious.

There are four major types of economically important parasitic flowering plants: broomrapes, dodders, witchweeds, and mistletoes.

Broomrapes

Broomrapes (Orobanche spp.) usually grow in clumps at the base of their host plants (Figure 6.4). They are root holoparasites. These parasites vary in color from whitish to pinkish, and they have very little green color because they do not produce chlorophyll and photosynthesize. There are many broomrape species, especially in warm, dry regions such as the Balkans, parts of Asia, the Middle East, and California. Host plants include brassica (crucifer) crops (e.g., cabbage), legumes, solanaceous crops (e.g., potato, tobacco, and tomato), and sunflower.

Figure 6.4. Broomrape (Orobanche ramosa) parasitizing tomato.

Dodders

Dodders (Cuscuta spp.) (Figure 6.5) are completely dependent on their host plants and are usually classified as stem holoparasites. They look like yellowish vines growing around the aboveground portion of the host, yet have no contact with the soil. Each stem produces numerous haustoria that connect to both the host xylem and phloem (Figure 6.6). After flowering, seeds fall to the ground, where they germinate or remain dormant until the following growing season. The tangle of yellowish stems reduces host plant growth and makes cultivation and harvesting difficult. Farmers gave these parasitic weeds names that reflected their annoyance: beggarweed, devil's guts, hellweed, and strangle tare. Dodders are sometimes called scaldweed, for the scalded appearance given to bean crops. Today, dodders are an economic problem mostly in perennial crops such as alfalfa and cranberries, where they reduce yield and interfere with harvest.

Figure 6.5. Dodder (Cuscuta pentagona) parasitizing herbaceous plants.

Figure 6.6. Dodder parasitizing a host plant (left). The invading haustoria (center and right) absorb water and nutrients from the phloem and xylem of the host.

Witchweeds

Witchweeds (Striga spp.) are deceivingly pretty green plants; they have colorful flowers above ground, but below ground they are aggressive parasites (Figure 6.7). Witchweeds appear to be typical plants that are green and photosynthesize, but they are actually root hemiparasites that emerge from the soil near the host plant. They absorb water and minerals via haustoria that penetrate host roots. High populations of witchweed can greatly reduce crop yields, especially where water and minerals are limited. Witchweeds are important pathogens of subsistence crops (e.g., corn, millet, rice, sorghum, and sugarcane) in Asia and Africa. They may be the most important agricultural pests in Africa. Witchweed was accidentally introduced into the United States (in North and South Carolina) in the 1950s.

Figure 6.7. A, Witchweed (Striga sp.) parasitizing the roots of a corn plant. B, Corn growing poorly because of witchweed infection. Note the witchweed plants between the corn rows.

Mistletoes

Mistletoes are parasites of trees. They are stem hemiparasites, connected primarily to the xylem. The leafy mistletoes (Phoradendron spp. in North America and Viscum spp. in Europe), used for Christmas decorations, are primarily parasites of hardwood trees and sometimes conifers. In the United States, leafy mistletoes are found where winters are not too cold, generally across the southern states and some coastal western states. Leafy mistletoes retain their leaves during the winter and are most easily noticed at this time of year, when their deciduous host trees are leafless. Mistletoes are dioecious, producing male and female reproductive structures on separate plants. The female plants of leafy mistletoes produce white berries, which are attractive to birds.

DID YOU KNOW?

In California, Luther Burbank introduced Viscum album (European mistletoe) around 1900, intending for it to be grown as a crop for sale at Christmas. V. album has become naturalized and has spread across a wide area, now over 100 square kilometers (about 40 square miles).

Like the leafy mistletoes, dwarf mistletoes (Arceuthobium spp.) are dioecious, but the plants are much smaller, vary in color, and have small leaves called scales (Figure 6.8). They are parasites of conifers. In the United States, they are found in the western forests and in the northern parts of the Great Lakes states and New England. Dwarf mistletoe seedlings are photosynthetic, but adult plants are almost completely dependent on their host plants via connections to both the xylem and the phloem. When these parasites invade large timber monocultures, heavy infections can occur. Dwarf mistletoes can distort the growth of conifers by causing witches’ brooms and reduce both the yield and the value of the timber.

Parasitic flowering plants that infect herbaceous plants (such as broomrape, dodder, and witchweed) cause symptoms similar to those caused by competition with free-living weeds. Heavily infected plants may be stunted, yellowed, and wilted, especially where water and minerals are limited. Leafy mistletoes often do little harm to their tree hosts, but dwarf mistletoes may reduce growth and cause deformities that affect the value of the harvested lumber. The parasitic flowering plants themselves are the pathogen signs.

Mistletoes Affecting Woody Plants

leafy mistletoes (Phoradendron spp., Viscum spp.)

- nondeciduous green leaves
- hemiparasites
- single seed in each white berry
- dioecious
- hosts: hardwoods (mostly) and conifers

dwarf mistletoes (Arceuthobium spp.)

- small plants with small leaves called scales
- hemiparasites, but produce only about 30% of their carbon through photosynthesis
- explosive dispersal of a single seed per fruit
- dioecious
- hosts: conifers

Figure 6.8. A female (fruiting) dwarf mistletoe (Arceuthobium campylopodum) parasitizing a shore pine in British Columbia, Canada.

Common Parasitic Plants Affecting Herbaceous Plants

broomrape (Orobanche spp.)

- root parasite
- holoparasite
- hosts: sunflower, tobacco, vegetable crops

dodder (Cuscuta spp.)

- stem parasite
- holoparasite
- wide host range

witchweed (Striga spp.)

- root parasite
- hemiparasite
- hosts: corn, legumes, rice, sorghum

How do parasitic flowering plants survive and spread?

As biotrophs (obligate parasites), parasitic flowering plants must either be attached to living hosts or survive as seeds. Ship ballast, packing materials, nursery stock, and potted plants can carry the seeds of parasitic plants. These are the same means of spread responsible for the movement of non-parasitic weed species and invasive plants.

Broomrape and witchweed produce tiny seeds that are less than half a millimeter in diameter. A single plant can produce thousands of long-lived seeds. Broomrape seeds have been reported to survive for 50 years. Witchweed seeds can survive for more than 20 years in the soil and do not germinate until they detect root exudates of a host plant. The tiny seeds have very little food reserves, so it is important for them to germinate only when a host plant root is nearby. The dustlike seeds can be blown by wind or carried by rain, irrigation water, and soil attached to tools and machinery and can contaminate seed lots of agronomic crops.

DISEASE CLASSIC

Witchweed

Hosts corn, legumes, rice, sorghum, sugarcane

Pathogen Striga species (root hemiparasites)

Symptoms and Signs The host plant symptoms are wilting and stunting and can also resemble those caused by nutrient deficiencies. Witchweed is a pretty, green plant with colorful flowers.

Disease Cycle The tiny seeds must be stimulated to germinate by root exudates of host plants. Witchweed absorbs water and minerals from the xylem of host roots. After establishment, green witchweed plants emerge from the soil near the host plant to photosynthesize and bloom, and each plant produces thousands of seeds. Yield losses caused by witchweed are greater where water and minerals are limited.

Management In subsistence agriculture, witchweed can be pulled by hand or destroyed by cultivation. Trap crops can be sacrificed after stimulating seed germination or soil can be treated with ethylene to stimulate germination. Some herbicides kill witchweed but not the host crop.

Significance Witchweed is a major parasite of subsistence crops in Africa and Asia. It was accidentally introduced into the United States in North and South Carolina, where it is under quarantine to prevent further spread.

Dodder seeds are about the same size as the seeds of many legumes that are common host plants, so they are easily harvested and planted along with the host seeds.

DISEASE CLASSIC

Dodder

Hosts many host plants; economically important in forage crops, such as alfalfa, and in cranberry

Pathogen Cuscuta species (stem holoparasites)

Symptoms and Signs Symptoms are wilting and stunting and can also resemble those caused by nutrient deficiencies. Dodder is a yellowish vine with small leaves called scales and small flowers.

Disease Cycle When a dodder seed germinates, the seedling grows upward, seeking the stem of a host plant. Once attached to a host, it loses contact with the ground and produces numerous haustoria, which absorb water and nutrients from the xylem and phloem of the host. The flowers produce fruit (capsules) with small seeds.

Management Small infestations of dodder should be pulled out before seed production. Larger areas may be plowed under or treated with herbicides, although the herbicides may also harm the host plants. Planting materials should be free of dodder seeds.

Significance Dodder is an economically significant problem mostly in perennial plantings. It is used in laboratories as a “living bridge” for experimental transmission of certain vascular pathogens, such as phytoplasmas and viruses.

Mistletoe seeds are coated with a tissue called viscin (Figure 6.9) that contains sticky, cellulosic fibrils that help the seeds attach to new infection sites. This tissue is reflected in the family name of mistletoes, Viscaceae. Female leafy mistletoe plants produce numerous white berries, each of which contains a single seed. Seeds may be ingested by birds, but are not digested, and pass through their bodies to be deposited at various sites. Seeds sometimes stick to birds and small mammals and are thereby carried to new sites. The female dwarf mistletoe plants produce a single seed in each fruit that is explosively discharged—as far as 16 meters (nearly 50 feet).

> Figure 6.9. Seeds of a leafy mistletoe (Phoradendron serotinum) floating in water. Note that the viscin coats are fully expanded and their cellulosic fibrils are visible.

Parasitic flowering plants survive and are dispersed as seeds. Broomrape and witchweed seeds are numerous and very tiny, whereas dodder seeds are larger. Contaminated seed lots, soil on machinery and plants, wind, and water can distribute the seeds of parasitic plants to new sites. Mistletoe seeds are coated with viscin to help them attach to new infection sites in trees.

How are diseases caused by parasitic flowering plants diagnosed?

Parasitic flowering plants can be identified in the same manner as weeds and other plants: by observing shoot and flower characteristics. As with other plants, identification to genus is easier than identification to species. All parasitic flowering plants are attached to their host plants, but it may not be immediately obvious that a plant is a parasite. For example, witchweed is a green plant with beautiful flowers, but it is connected to the root system of its host plant below ground. In contrast, holoparasites are not green and have very small leaves.

Parasitic flowering plants are diagnosed by establishing their connections to host plants and identifying the parasite by morphological features.

How are parasitic flowering plants managed?

Management practices for parasitic flowering plants are similar to those used for free-living weeds.

Avoidance

It is sometimes possible to avoid parasitic flowering plants by choosing locations where parasite populations are low. For example, planting sites for conifer timber production can be evaluated for the potential severity of infection by dwarf mistletoes.

Exclusion

Careful inspection of imported plant and shipping materials may reveal living parasitic flowering plants, but it is difficult to monitor and exclude seeds, which may be very tiny. Seed for planting should be inspected for seeds of parasitic plants such as dodder.

Witchweed could potentially spread throughout the southeastern and midwestern corn-, rice-, and sorghum-growing areas of the United States. A quarantine is in effect in North and South Carolina to limit the movement of soil, machinery, crops, and other materials that may carry witch-weed seeds.

DID YOU KNOW?

Five genera of parasitic plants are on the U.S. Federal Noxious Weed List: Aeginetia, Alectra, Cuscuta, Orobanche, and Striga. Noxious weeds are parasitic or foreign plants that are new to or not widely prevalent in the United States and can directly harm plants, livestock, public health, etc.

Eradication

In subsistence agriculture, hand-pulling of parasitic plants, such as broomrape and witchweed, especially before they produce seeds, is common. In minor invasions of flower beds or landscapes, dodder should be removed before it produces seeds. Physical removal and herbicide treatments are sometimes used to destroy dodder in larger areas, such as alfalfa fields and cranberry plantings, but it is difficult to find herbicides that selectively kill the parasite without harming the host.

DID YOU KNOW?

One creative management strategy for parasitic flowering plants is to eat them! Broom-rape stems are sometimes cooked and eaten like asparagus. Dodder seeds are sometimes eaten in the southwestern United States. Some parasitic flowering plants are harvested for use as herbal medicines.

Considerable research has been done to understand how root exudates stimulate seed germination in witchweed. Unfortunately, even a single witchweed plant can produce thousands of tiny seeds (Figure 6.10) that can survive in the soil until stimulated to germinate by a host plant. Various trap crops have be used to reduce the seed population in the soil. Witchweed hosts are planted to stimulate seed germination and then are sacrificed by plowing them under before the witchweed produces new seeds. Decoy crops stimulate seed germination but are not susceptible to witchweed infection. Without host plants, the new witchweed plants die. Soil can also be treated with ethylene, although the treatment is costly. Ethylene has been found to stimulate germination of up to 90% of witchweed seeds in some areas. The young witchweed plants can then be destroyed before a crop is planted. All three of these methods reduce the witchweed seed population in the soil, so a susceptible crop can be planted.

Resistance genes for nonselective herbicides such as glyphosate have been engineered into certain crop plants. Herbicides applied to these plants eradicate both free-living weeds and parasitic flowering plants without injuring the crop.

DID YOU KNOW?

A single witchweed plant (Striga sp.) can produce more than 50,000 seeds, which can remain viable in soil for up to 20 years.

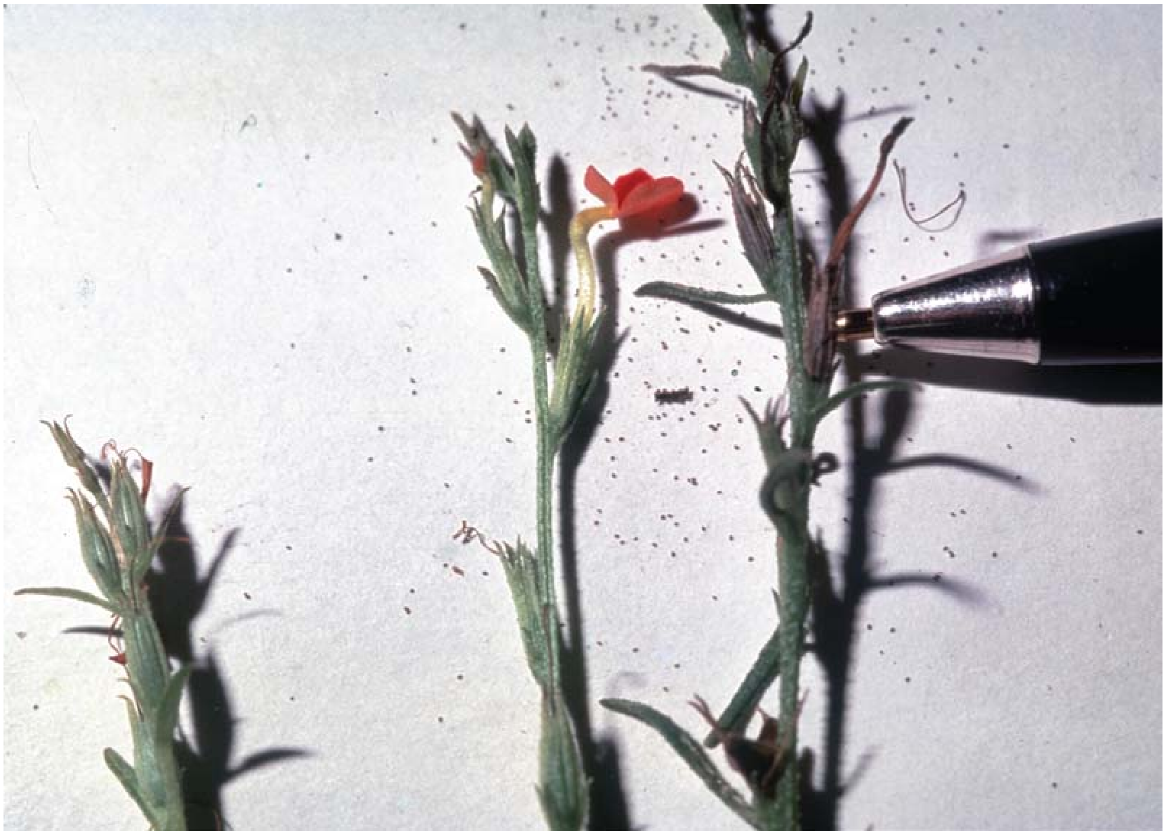

Figure 6.10. Witchweed (Striga sp.) seeds, scattered on the paper beneath the plants, are numerous and tiny.

Leafy mistletoes can be cut from tree branches, but they often grow back because the mistletoe tissues invade widely along the length of the branch. Selective pruning of small branches when infections are first noticed is most effective. If only a small amount of mistletoe is present, it may cause little real harm to the tree. Removal of female plants with berries may help reduce the spread of leafy mistletoe in landscapes. Mistletoe with berries should not be used for outdoor Christmas decorations in areas where mistletoe can survive, because birds will be attracted to the berries and may spread the seeds to nearby trees.

Managing dwarf mistletoe infections of large conifer timberlands is more challenging. Dwarf mistletoes are important factors in modern forestry management, affecting thinning, replanting (clear-cutting versus selective cutting), and fire control practices. Although clear-cutting increases soil erosion, it also provides a clean site for planting young trees. When young trees are planted among older, infected specimens, invasion by dwarf mistletoe may occur more quickly. Fire in the western forests of North America has long been an important natural means of pest and pathogen control. Disruptions in the natural fire cycles have resulted in increases in a number of forest pests and pathogens, including dwarf mistletoes.

DID YOU KNOW?

Dwarf mistletoes cause direct volume loss (growth loss plus mortality) of 3.3 billion board feet (a measure of wood volume) per year in the western United States. That's

- enough wood to build between 243,000 and 1,000,000 houses a year!
- about five times the total annual timber harvest of the entire state of New York!
- roughly one-fifth of the annual timber harvest from the contiguous United States west of the Mississippi!

Source: James Worrall

DISEASE CLASSIC

Dwarf Mistletoe

Hosts conifers

Pathogen Arceuthobium species (conifer hemiparasites)

Symptoms and Signs Heavily infected conifers may be stunted and malformed with witches' brooms. Dwarf mistletoe plants are generally small and are dioecious with inconspicuous flowers.

Disease Cycle Female plants explosively expel their seeds, which are coated with viscin to help them attach to hosts. The germinating seed invades through young branch tissue and establishes a complex structure, called an endophyte, deep in the host plant tissue before the shoots emerge. The mistletoe obtains nearly all of its food and water from the host via haustoria. In nature, dwarf mistletoes may require 3 to 10 years to complete their life cycle.

Management Early infections of dwarf mistletoes can sometimes be pruned from individual conifers. Management in a forest monoculture is based on the planting of new trees where dwarf mistletoe populations are low and eradication of the parasite through the use of controlled fire.

Significance Dwarf mistletoe is a significant conifer timber parasite that reduces yield and lumber quality by causing witches’ brooms and malformed trees.

Plant Disease Profiles: Dwarf mistletoe

Like their host plants, parasitic flowering plants are affected by pests and pathogens. Researchers are investigating pathogens, especially fungi, that infect the parasitic plants but not their hosts. These pathogens are potential biological control agents, but no commercial products are yet available.

Protection

Like those affected by weed problems, plants infected by parasitic flowering plants can tolerate the pathogens better when other growth factors, such as soil quality, water, and nutrients, are not limiting. Some cultivars and species are more resistant to or tolerant of certain parasitic flowering plants, giving hope for the selection of genetic resistance, especially in areas where the parasites are endemic. For example, identifying host genotypes that provide resistance to witchweed is a major focus of witchweed research. It might be possible to pyramid several resistance genes into specific crop cultivars to provide stable and cost-effective resistance. Recent research with broomrapes suggests that gene silencing (see Chapter 9) of parasite-specific genes might also increase host plant resistance.

Parasitic flowering plants of herbaceous plants are managed like other weeds. Care is taken to avoid introducing the seeds into soil, water, or planting materials. Sometimes parasitic plants can be removed by hand or killed by herbicides, preferably before they produce new seeds. Mistletoes may be pruned from individual trees. Controlled burning and selective cutting of heavily infected trees reduce dwarf mistletoe populations in conifer timberlands. Host cultivars with resistance or tolerance to certain parasitic plants can be selected.

Management Strategies for Parasitic Flowering Plants

Avoidance

- Choose planting sites where parasite populations are low.

Exclusion

- Impose quarantines (local and international).

Eradication

- Use decoy or trap crops.
- Apply herbicides.
- Remove plants.

Protection

- Plant resistant or tolerant species or cultivars.

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Questions

1. How are parasitic flowering plants different from nonparasitic weed species?
2. How do parasitic flowering plants absorb water, minerals, and sometimes photosynthates from their host plants? In what other group(s) of pathogens is this same word used to describe a specialized structure used for nutrient absorption?
3. Parasitic flowering plants may be primarily xylem parasites (hemiparasites) or they may parasitize both the xylem and phloem (holoparasites) of their hosts. What characteristics would you expect a hemiparasite to have? What characteristics would you expect a holoparasite to have?
4. What is the basis for the name “witchweed”? What are several aspects of witchweed that make it a significant agricultural pest? Do you think it will be eradicated or at least contained in the United States, or will it continue to expand its range in North America?
5. Dwarf mistletoe became a more important conifer forest pest in the twentieth century. What changes in forest management caused this development?
6. Why did ancient people ascribe magical properties to the so-called Christmas mistletoe?

Challenge Questions

1. How is management of parasitic flowering plants on agronomic and ornamental plants similar to and different from management of weed species?
2. What are some evolutionary advantages and disadvantages to being a parasitic plant?
3. Some parasitic flowering plants (and other angiosperms) are dioecious. What does this mean? What possible advantages and disadvantages might this have for a plant?
4. What other kinds of pests and pathogens cause witches’ brooms to form on plants?

Supplementary Resources

Illustrated Glossary

Special Topic

Introduction to parasitic flowering plants

Plant Disease Profile

Dwarf mistletoe

WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

avoidance biotroph

decoy crop

dioecious

eradication

ethylene

exclusion

haustorium (pl. haustoria)

hemiparasite

holoparasite

obligate parasite

protection

trap crop

witches’ broom

Exercise

Exercise 6.1. Identify the parasitic flowering plants shown in the images presented.

Internet Resources

APS Resource

What does mistletoe have to do with Christmas?

Recommended Website

The Parasitic Plant Connection

Internet Research

Internet Research Exercise 6.1. Find a fact sheet or disease profile describing a parasitic flowering plant that affects a plant of interest to you. How do you identify the parasite? Does the parasite exist where you live? What management methods are recommended? Are resistant or tolerant cultivars of an economically important host available?

Internet Research Exercise 6.2. Find a fact sheet or disease profile describing a dwarf mistletoe species that exists in the eastern part of the United States and a species that exists in the western mountain states. What conifer species are parasitized by each pathogen? What management practices are recommended in each area? Could dwarf mistletoe be managed in either part of the United States by rotating timber species?

CHAPTER 7

What Are the Causes of Plant Diseases?

► Abiotic Factors

IN THIS CHAPTER:

- ► **What are abiotic factors?**
- ► **What symptoms do abiotic factors cause?**
- ► **How are abiotic diseases diagnosed?**
- ► **How are abiotic diseases managed?**

What are abiotic factors?

Discussions of plant diseases tend to focus on infectious diseases, caused by fungi, bacteria, nematodes, viruses, and other biotic pathogens. There is another group of problems, caused by **abiotic**, or nonliving, factors. Some of these meet the definition of "disease," according to which the plant is harmed progressively over time. Others affect the health of the plant in a more immediate manner, often leading to **injury**. Examples of plant injuries include lightning strikes and hail damage (Figure 7.1).

Some Causes of Abiotic Plant Diseases

- **environmental factors, extremes in**

- temperature
- moisture
- light
- nutrients (mineral elements)
- pH

chemicals

- air pollutants (e.g., ozone, sulfur dioxide, nitrogen oxide)
- pest-control products, especially herbicides, if misapplied
- deicing salt

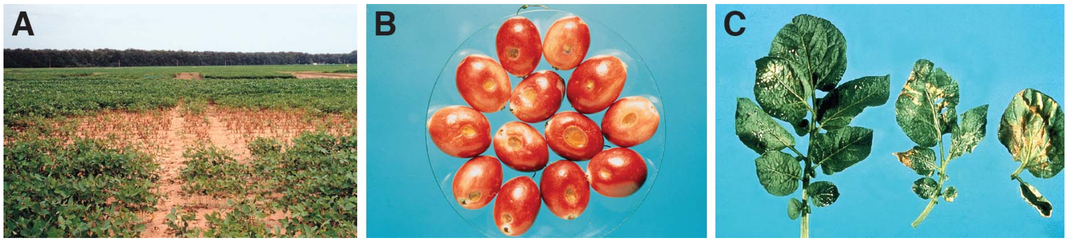

Figure 7.1. Injury caused by A, lightning in a soybean field; B, hail on cranberries; and C, wind in potato plants.

Plant diseases caused by abiotic factors have been called noninfectious diseases, physiological diseases, or environmental diseases. It is difficult to find just the right term for this wide variety of factors, but biotic pathogens are not involved in abiotic diseases, and therefore, these diseases are not infectious or transmissible. Abiotic factors may, however, increase or decrease a plant's susceptibility to biotic pathogens and its sensitivity to other abiotic factors.

Abiotic problems are of particular concern when plants are grown in pots indoors. Plants growing in homes, offices, and shopping malls may be exposed to fewer biotic pathogens than are outdoor plants, but the growing conditions may be less than optimal because of artificial light sources, uneven watering, and a restricted environment for root growth.

In this chapter, we consider the various abiotic factors that cause plant diseases by reducing growth or yield or detracting from the desired appearance of plants. Certain plant species have adapted to grow and reproduce under a relatively narrow range of environmental conditions. Abiotic diseases often result when conditions are outside this range.

In general, the diseases caused by abiotic factors can be grouped into two categories: those caused by excesses or deficiencies in environmental factors, such as temperature, moisture, light, and nutrients, and those caused by exposure to harmful chemicals, such as air pollutants, herbicides, and salt.

Environmental Factors in Abiotic Disease

Disease or injury can result when any of several environmental conditions are outside of a plant's normal range of requirements. The abiotic factors most commonly involved are temperature, moisture, light, wind, mineral elements, and soil **pH**.

What is soil pH?

Soil pH is a measure of the concentration of hydrogen ions in the soil, which is a measure of the soil's acidity. The pH scale goes from 1 to 14, with 1 being the most acidic and 14 being the most alkaline (or basic). An increase or decrease of one number on the pH scale represents a tenfold change in acidity or alkalinity. The "ideal" pH for soil is between 6.2 and 6.8 because nutrients (mineral elements) are most available to plants in that range.

TEMPERATURE

High temperatures occasionally injure plants, especially when accompanied by UV radiation or moisture stress. It is much more common, however, for plants to be exposed to temperatures below their optimal range for growth and development. Low temperatures damage plants primarily by inducing the formation of intercellular and intracellular ice crystals. The formation of ice crystals is influenced by the presence of some species of epiphytic bacteria that act as catalysts for ice crystal formation. When the cytoplasm freezes, the cell membrane ruptures, releasing the cell contents and resulting in cell death.

The extent of damage to plants by low temperatures is determined by the lowest temperature reached, the rate of temperature change, and the duration of the cold spell. Tropical plants may suffer damage from cool temperatures well above freezing (below 16–18°C, or 60–65°F). Plants in colder climates protect themselves from freezing in several ways: by completing their life cycles and having dormant seeds that survive the cold, by retaining belowground tissues and regrowing aboveground tissues the following year, or by gradually shutting down many metabolic processes during the fall (a process called hardening-off, or cold-hardening).

► **DID YOU KNOW?**

Florida is periodically hit by damaging freezes that destroy the winter vegetable crops (green beans, peppers, squash, tomatoes) that provide about one-third of the warm-season vegetables purchased by people in snowy northern states. The freeze of 1997 struck central and southern Florida and caught most growers by surprise, leaving them with $300 million in losses.

Some cold injury is due to frost crystals that form from the freezing of water outside the plant cells. For example, frost on turfgrass does not usually kill the leaves, but it makes the leaf blades brittle and subject to breakage if walked on. Footshaped areas of brown turf develop after this injury.

More severe injury occurs when the liquid inside the plant cells freezes and ruptures the cell membranes. Most losses due to cold temperatures occur in late spring when a freeze damages new growth and flowers or in fall when an early freeze harms foliage, fruits, or vegetables that are not yet mature or hardened-off for the winter. Stored fruits and vegetables also are subject to cold injury, which can reduce quality and value.

MOISTURE

Both insufficient and excessive amounts of water can harm plants. Low soil moisture, often called **drought**, is probably responsible for more plant damage than any other abiotic factor (Figure 7.2A). Water also is not available to plants from frozen soils in winter. If plants retain their leaves in the winter and continue to lose water by transpiration caused by dry winter winds and sunshine, drought effects may be observed. Excessive soil moisture, often in the form of flooding, also can lead to plant damage and death (Figure 7.2B). This is probably the most common cause of death of potted plants that do not have sufficient drainage. Excessive soil moisture deprives roots and other underground structures of oxygen. The root cells die, and the roots may be invaded by pathogens and saprophytes in the soil. In addition, anaerobic conditions favor the activity of denitrifying bacteria. These bacteria convert nitrates to the gaseous form of nitrogen, N_2, and nitrogen deficiency can occur relatively quickly in plants in flooded or poorly drained soils. In addition, various toxic substances can accumulate over time in flooded soils, including hydrogen sulfide, methane, organic acids, and reduced forms of metal ions.

► DID YOU KNOW?

Winterburn of plants is not caused by heat but by the dry winter winds blowing across leaves that cannot replace their moisture because the ground is frozen. This injury is sometimes not evident until the following summer, when plants are exposed to drought.

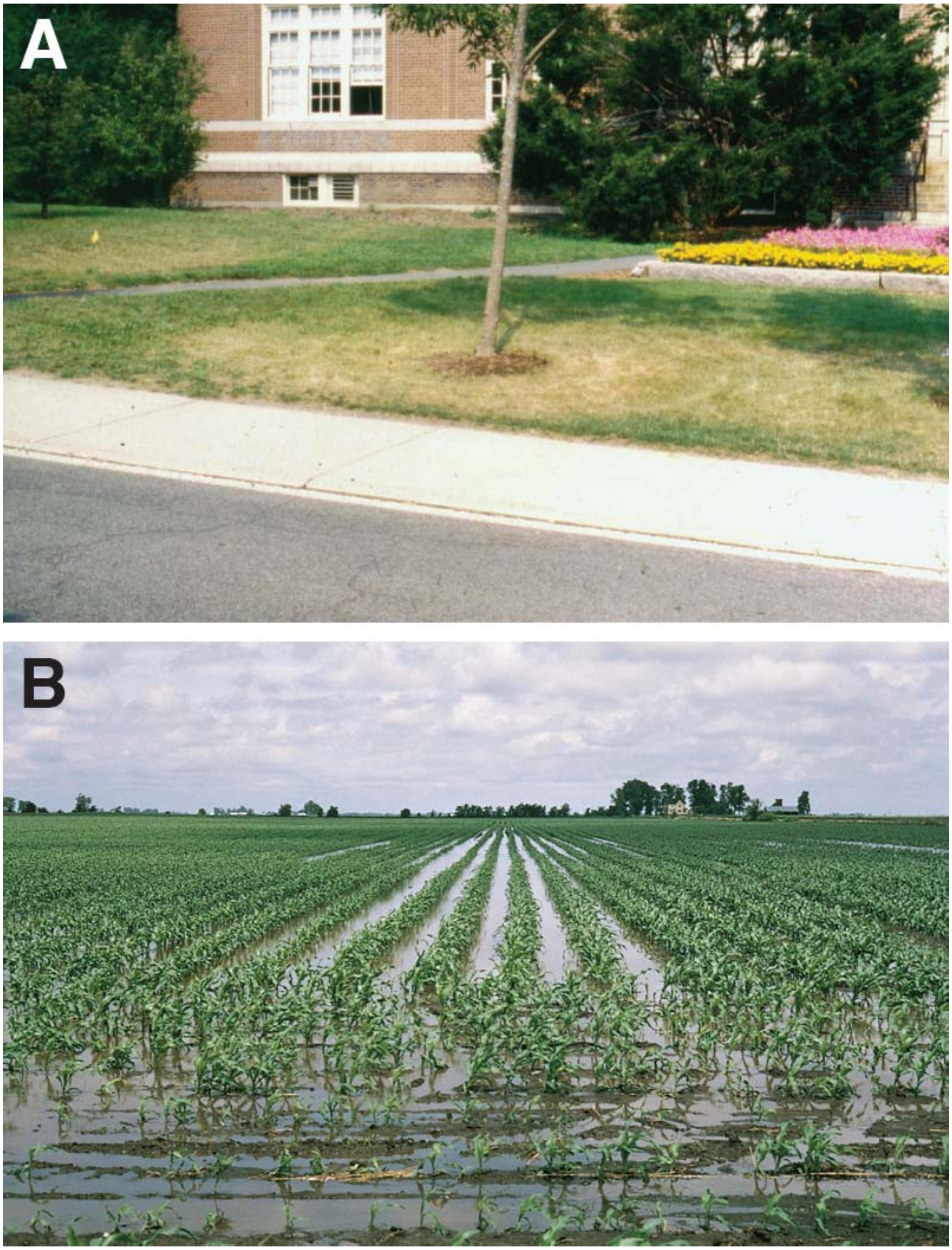

Figure 7.2. A, Drought stress symptoms on turfgrass. B, Flooding injury on corn.

LIGHT

Plants also can be damaged by either excessive or insufficient amounts of light. Insufficient light is more common for plants grown in building interiors, homes, and greenhouses, especially in the winter months. The effects of light can often be noticed when a plant is moved from one area to another where the quantity of light is significantly different. For example, plants that have been hanging on a porch throughout the summer may show symptoms of reduced light supply when brought inside for the winter. **Sunscald**, or **sunburn**, is an injury caused by intense radiation from the sun or the heat that it creates in plant tissues. Fruits or vegetables may be damaged when they are suddenly exposed to full sun after a windy storm that disrupts the leaf canopy (Figure 7.3). Injury is common when young plants grown indoors are transplanted into the full sunlight of an outdoor garden. Intense sunlight also can injure the trunks of young trees.

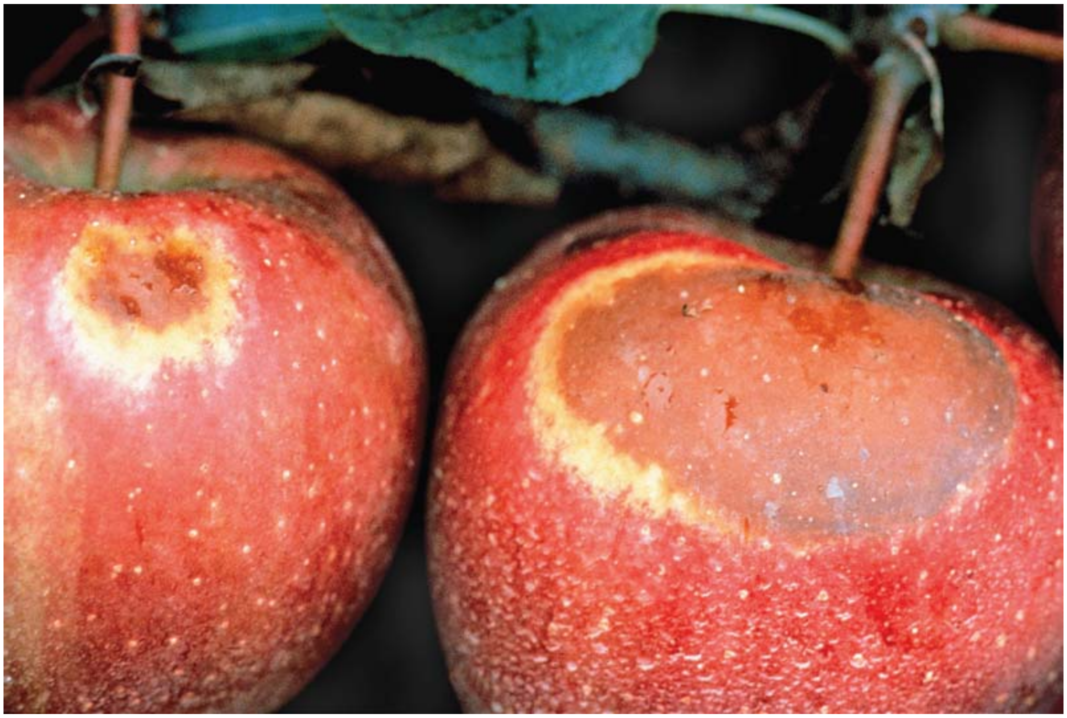

Figure 7.3. Sunburn on Delicious apples.

MECHANICAL INJURY

Extreme weather can injure plants. Hail can destroy fruit crops if it occurs when the fruits are young. Excessive winds can cause plants to tip over, or **lodge**, and can break branches of woody plants. Lightning strikes can injure tall trees or can leave mysterious circular patches of injured plants in open areas with few trees. Animals, such as rodents and deer, injure trees and shrubs during feeding. Human activities, such as construction and careless use of weed whackers and lawn mowers, can injure root systems and damage trunks of trees. The most damaging human activity, however, is placement of plants in an environment that is not conducive to good growth.

► **DID YOU KNOW?**

When apples or pears are injured by hail early in the season, suberization (formation of cork cells) heals the wounds. Long after the original injury, large scars or deformed areas develop as the fruits mature, making them unmarketable.

NUTRIENT IMBALANCE

All plants require 20 essential elements for growth and reproduction. Carbon and oxygen are supplied in the air; the rest are supplied in water or in soil. Some elements are required in relatively large quantities and are called **macronutrients**. They include nitrogen (N), phosphorus (P), potassium (K), calcium (Ca), magnesium (Mg), and sulfur (S). Some of these are important structural components of plant cells; others perform regulatory functions. If a sufficient quantity of any of these elements is not present in the soil, the plant will suffer a **nutrient deficiency** disease (Figure 7.4). Sometimes an adequate supply of an element is present in the soil, but because of other factors (such as soil pH or the balance of nutrients present), plants are unable to absorb adequate amounts of the element, and a nutrient deficiency disease results.

Important Plant Nutrients

Macronutrients	Micronutrients
nitrogen (N)	boron (B)
phosphorus (P)	chlorine (Cl)
potassium (K)	copper (Cu)
calcium (Ca)	iron (Fe)
magnesium (Mg)	manganese (Mn)
sulfur (S)	molybdenum (Mo)
	zinc (Zn)

Macronutrients are required in higher quantities than micronutrients.

Micronutrients are necessary for plant growth but may become toxic over a relatively narrow range of concentrations.

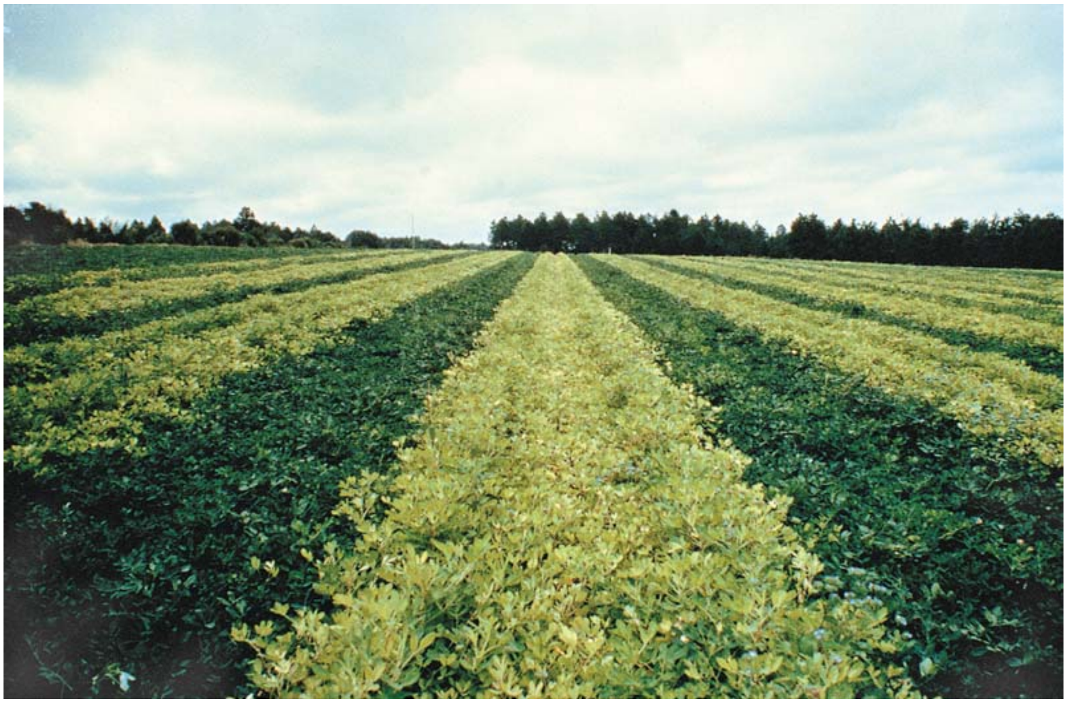

Figure 7.4. Nitrogen deficiency symptoms on peanut.

Other elements are required by plants in relatively small quantities and are therefore called **micronutrients**. They include boron (B), chlorine (Cl), copper (Cu), iron (Fe), manganese (Mn), molybdenum (Mo), and zinc (Zn). Several of these elements are constituents of enzyme complexes or important parts of plant components, such as the iron that is contained in chlorophyll. Most natural soils contain sufficient amounts of micronutrients, although some specific plants may have greater-than-normal demands for some of these elements. Micronutrient deficiencies are sometimes observed in plants grown in containers for long periods. Plants also may suffer from an excess of these elements in their environment, resulting in a **nutrient toxicity**. Soil pH may cause micronutrients to become more or less available, because pH affects their solubility in water. Because the range of concentrations that is appropriate for a plant is much narrower for micronutrients than for macronutrients, soil pH may have a bigger impact on micronutrient deficiencies and toxicities than on macronutrient disorders. There are many complex interactions among the elements required by plants, and a deficiency or excess of one element can influence the availability and activity of others, sometimes with catastrophic consequences.

Exposure to Harmful Chemicals

The second major category of abiotic plant diseases results from exposure to chemicals that are not normally present in the plant's environment. The most common factors involved are air pollutants, pesticides such as herbicides, and salts.

POLLUTION

Air pollution is a general term referring to the contamination of the atmosphere with products from human activities. Pollutants sometimes cause obvious symptoms, but they also can reduce yields when no symptoms are visible. Three "modern" activities of people have caused most of the increase in major pollutants in our air: the burning of fossil fuels, the driving of motor vehicles, and the manufacturing that came with the Industrial Revolution. It seems unlikely that we are about to give up any of these activities, so we need to learn what problems they are causing and what, if anything, we can do to reduce them.

Principal Sources of Important Air Pollutants and Symptoms of Air Pollution Injury

ozone

internal combustion engine exhaust + sunlight

flecking (upper leaf), stippling, mottling, tipburn (conifers), stunting, defoliation

peroxyacetyl nitrate (PAN)

internal combustion engine exhaust + sunlight
bleaching, bronzing (lower sides of leaves)

sulfur dioxide
industrial processes, coal burning
interveinal necrosis (broadleaf plants), tip necrosis (conifers)

nitrogen oxide
internal combustion engines, industrial processes
bronze- or copper-colored necrosis of leaves

acid precipitation
sulfur dioxide or nitrogen oxide + water
chlorosis and necrosis of leaves, stunting

hydrogen fluoride
industrial processes (e.g., aluminum smelters, brick production)
marginal and tip chlorosis and necrosis of leaves

ethylene
internal combustion engines, industrial processes
defoliation, senescence, leaf deformation, fruit drop

Common air pollutants include ozone, sulfur dioxide, nitrogen oxide, peroxyacetyl nitrate (PAN), hydrogen fluoride, and ethylene. The most important air pollutants in terms of plant disease are ozone, sulfur dioxide, and nitrogen oxide. **Ozone** (O_3) and PAN are called **secondary pollutants** because they are produced by the interaction of sunlight with other pollutants in the exhaust from internal combustion engines. Levels of ozone are higher during the day and lower at night because ozone is produced by **photochemical reactions** that require sunlight. Warm conditions favor these reactions, so ozone levels are higher in the summer and lower in the winter (Figure 7.5).

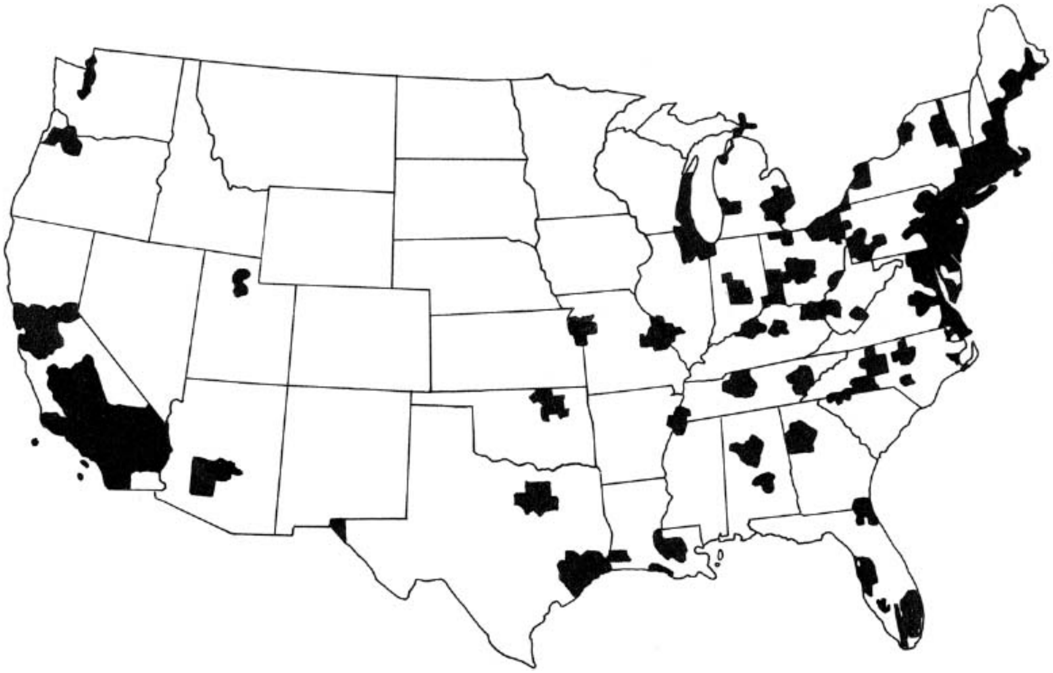

Figure 7.5. U.S. regions that violated federal air-quality standards for ozone more than once a year for three years in a row.

Ozone is the most significant air pollutant worldwide in terms of damage to plants. Leaves of plants exposed for even a few hours to ozone concentrations as low as 0.1–0.5 ppm can be damaged, and yield losses of more than 50% have been reported, with dicot species being more sensitive than monocot species. Yield losses may be due to a single (**acute**) exposure to a high concentration of ozone or to longer-term (**chronic**) exposure to lower concentrations. Alfalfa, beans, cereals (including corn), citrus, petunia, pine, spinach, and tobacco are among the most ozone-sensitive plants. Annual losses to the U.S. economy caused by ozone are measured in billions of dollars, but often these losses are not recorded because the symptoms of ozone damage are difficult to recognize and often resemble natural senescence. The ozone layer that helps protect us from ultraviolet radiation is not harmful to plants because it is far above the environment in which plants live, ranging from about 15 to 35 kilometers (10 to 20 miles) above the earth's surface.

Peroxyacetyl nitrate (PAN), like ozone, is a product of a photochemical reaction in the atmosphere with exhaust products of the internal combustion engine. Ozone, PAN, and smoke can combine to form what is commonly called photochemical "smog."

Internal combustion engines and industrial sources, such as power plants and refineries, also produce **sulfur dioxide** (SO_2) and **nitrogen oxide** (NO_x). These are called **primary pollutants** because they are released directly into the atmosphere from their sources. Less than 3 ppm of either of these pollutants can cause toxic effects in many different kinds of plants, including field crops, ornamentals, and trees (Figure 7.6). In addition, these compounds combine with water in the air to produce sulfuric acid and nitric acid, resulting in a secondary pollutant called "acid precipitation," which can be fog, rain, or snow with a pH lower than 5 (Figure 7.7).

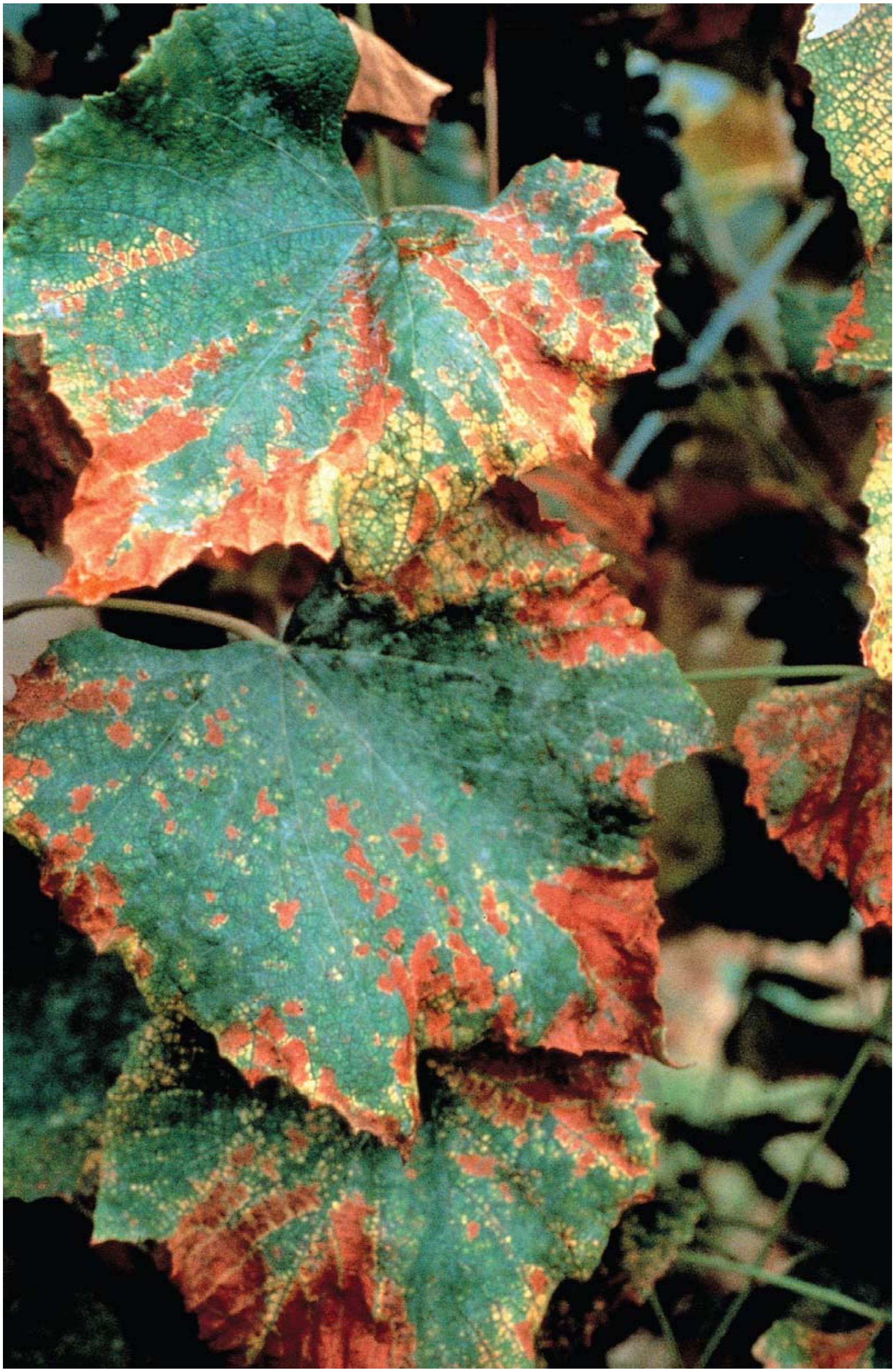

Figure 7.6. Marginal and interveinal chlorosis and necrosis on grape leaves caused by sulfur dioxide.

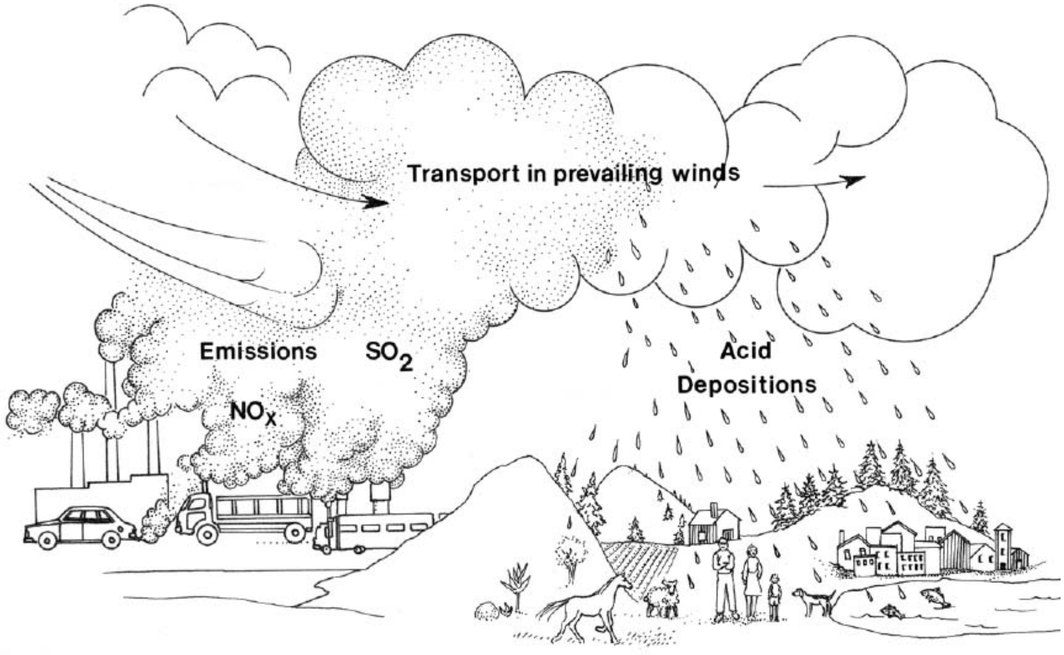

Figure 7.7. Origins of acid precipitation.

Acid precipitation has received a lot of attention as a possible cause of **forest decline**, a progressive deterioration of forests over many years (Figure 7.8). The effects of acid precipitation on forests may be either direct or indirect. For example, acid deposition on leaves may cause leaching, or loss, of minerals, or the lower pH of the precipitation could lower the pH of the soil, which in turn could change the concentrations and availabilities of different nutrients in the soil. In some areas, forest decline is directly attributable to acid precipitation. In other areas, the situation is much more complex, and acid precipitation is probably only one of several contributing factors.

Figure 7.8. Forest decline of red spruce. A, Red spruce mortality, Crawford Notch, White Mountain National Forest, NH. B, Dead red spruce, Santanoni Peak, Adirondack Park, NY.

Other air pollutants that can damage plants include chlorine, ethylene, hydrogen chloride, and hydrogen fluoride. **Ethylene** is a plant growth regulator produced by plants, and it also can be produced when fossil fuels are burned. Chlorine, hydrogen chloride, and hydrogen fluoride are products of industrial processing. **Hydrogen fluoride** is a common pollutant from volcanoes and several industries, such as brick factories, aluminum smelters, and phosphate fertilizer factories. It is one of the few air pollutants that accumulates in plant tissues (Figure 7.9). Animals grazing near these factories may develop fluorosis, characterized by weakened teeth and bones, from ingestion of fluoride-contaminated forage plants.

Figure 7.9. Marginal and interveinal necrosis on grape leaves caused by hydrogen fluoride emitted from an aluminum-processing plant.

Some air pollutants play a role in the “greenhouse effect,” which is the increase in the air temperature at the earth’s surface due to radiation that is trapped on the surface and then reradiated into the atmosphere. Surface pollutants, including O_3 and NO_x, trap this reradiation and thus contribute to global warming. Several studies of temperature data collected since the early 1900s indicate that, although daytime temperatures have decreased slightly, nighttime temperatures have increased. The effects of these temperature changes on plant diseases need to be examined. Even small changes in temperature can affect frost-free dates, average temperatures and temperature extremes during the growing season, rainfall patterns, and extreme weather events.

HERBICIDES

Herbicides (weed killers) are used to manage undesired plant species, and they sometimes damage desired plants (Figure 7.10). Some common reasons for herbicide damage are **misapplication**, **drift**, **movement in water**, and **persistence in soil**. Occasionally, there are even concentrated spills of herbicides. Misapplication occurs when an applicator uses an herbicide not registered on a particular plant, applies the incorrect dose, uses an inappropriate application method, uses an inappropriate tank mixture of other chemicals, uses a contaminated spray tank, or applies at the incorrect time. Drift occurs during application when the herbicide is blown by the wind or volatilizes and moves to nontarget plants. This is sometimes referred to as “chemical trespass.” An herbicide also may move in water into an adjacent field of nontarget plants. When environmental conditions do not favor herbicide degradation, the herbicide may persist in the soil until the next cropping season, when it may damage a susceptible crop. Drought can increase the persistence of some herbicides because of the reduced activity of microorganisms that normally degrade them. Herbicide damage is especially significant when it affects ornamental plants or fresh food crops, the appearance of which is important.

Figure 7.10. Damage from herbicides due to (A) misapplication of glyphosate on lettuce and (B) drift of paraquat on beans.

DEICING SALT

In northern areas, salt used to melt ice and snow on roads, sidewalks, and parking lots can injure or kill nearby plants. Most deicing salts contain the chloride ion (Cl^-), which is toxic to plants when taken up by roots or sprayed on foliage. Turfgrass exposed to runoff contaminated with salt is often killed along the edges of roads and sidewalks. Conifers along highways may turn brown because they are exposed to salt spray from traffic. Low-growing evergreen landscape plants also may exhibit salt toxicity from traffic spray or soil contamination. Salts also can accumulate in potting media when irrigation does not flush them through the root zone, as usually occurs in outdoor soils. In coastal areas, vegetation is sometimes damaged by salt carried by winds off the ocean.

► **DID YOU KNOW?**

Eight to twelve million tons of rock salt (NaCl) is applied to roads in the United States each winter. Deicing salt can enter the soil, ground-water, and surface water directly, from snow-melt runoff, or from windborne spray. The salt remains in solution in surface waters, and its accumulation and persistence in watersheds pose risks to aquatic ecosystems and to water quality.

Abiotic factors that cause disease in plants can be grouped into two categories: environmental factors (e.g., temperature, moisture, light, wind, mineral elements, and soil pH) and chemicals (e.g., primary and secondary air pollutants, herbicides, and salt). Both excesses and deficiencies of environmental factors can cause disease. Some abiotic factors are natural; others are the result of human activities.

► **DID YOU KNOW?**

In the 1980s, people became concerned that major forests, such as Germany’s famous Black Forest, were dying. This “forest decline” was originally thought to be caused by air pollution. Now forest decline is considered to be the result of many factors, including diseases, insects, and pollution, as well as part of a natural cycle of renewal in forests.

What symptoms do abiotic factors cause?

Symptoms caused by abiotic factors can be similar or identical to those caused by biotic pathogens (Figures 7.11 and 7.12D). Symptoms may be obvious when exposure to the abiotic factor is acute (i.e., a high concentration for a short period), but chronic, low-level exposure is more common for many of these problems and more difficult to detect. It also is not uncommon for more than one abiotic factor to influence a plant’s health. In some cases, we observe additive symptom effects from each factor. In other situations, the factors interact in a synergistic way, causing more injury than we would expect from the combined impacts of the factors, or in a negative way, with one factor canceling out some of the effects of another. The interactions of abiotic factors with biotic pathogens are similarly complex and can be additive, synergistic, or negative.

► DID YOU KNOW?

The leaves of African violet (*Saintpaulia ionantha*) are very sensitive to cold water. If cold water is applied to them, they will develop bright yellow rings, which can be confused with symptoms of virus infection.

Figure 7.11. Ring spots on African violet leaves injured by cold water, resembling symptoms of virus infection.

Symptoms Due to Environmental Factors

Environmental factors can cause a range of symptoms in affected plants (Figure 7.12A–E). Plant tissues damaged by exposure to low temperatures often appear water-soaked and then become necrotic. Severely damaged tissues may wilt or become flaccid. Tips or edges of leaves or vascular tissue inside buds or twigs may become discolored. Frosts and freezing temperatures can kill leaf or flower buds, kill entire herbaceous plants, and crack roots or trunks of woody plants.

Figure 7.12. Symptoms caused by environmental factors. A, White drupelet disorder of red raspberry caused by high temperatures and ultraviolet radiation. B, Freeze injury on orange fruit. C, Edema on geranium caused by high relative humidity, high soil temperature, and overwatering. D, Wilt on poinsettia caused by lack of water or by root-infecting pathogens. E, Sunscald on pepper.

Drought can cause plants to have pale leaves, as if malnourished, to wilt, to produce fewer leaves and fruits than normal, and even to die. Plants can often survive short periods of water shortage (**temporary wilt**), but even perennial plants are damaged by long periods of drought. **Scorch** on leaves, a marginal necrosis, is common during droughts. **Winterburn** (Figure 7.13) is the name for scorch and necrosis of plants that retain their leaves in the winter but cannot take up water from frozen soils. Woody plants with winterburn may produce less annual growth and have smaller leaves, which are dropped prematurely. These same symptoms can be caused by vascular wilt pathogens (fungi and bacteria) and root pathogens (fungi, bacteria, and nematodes).

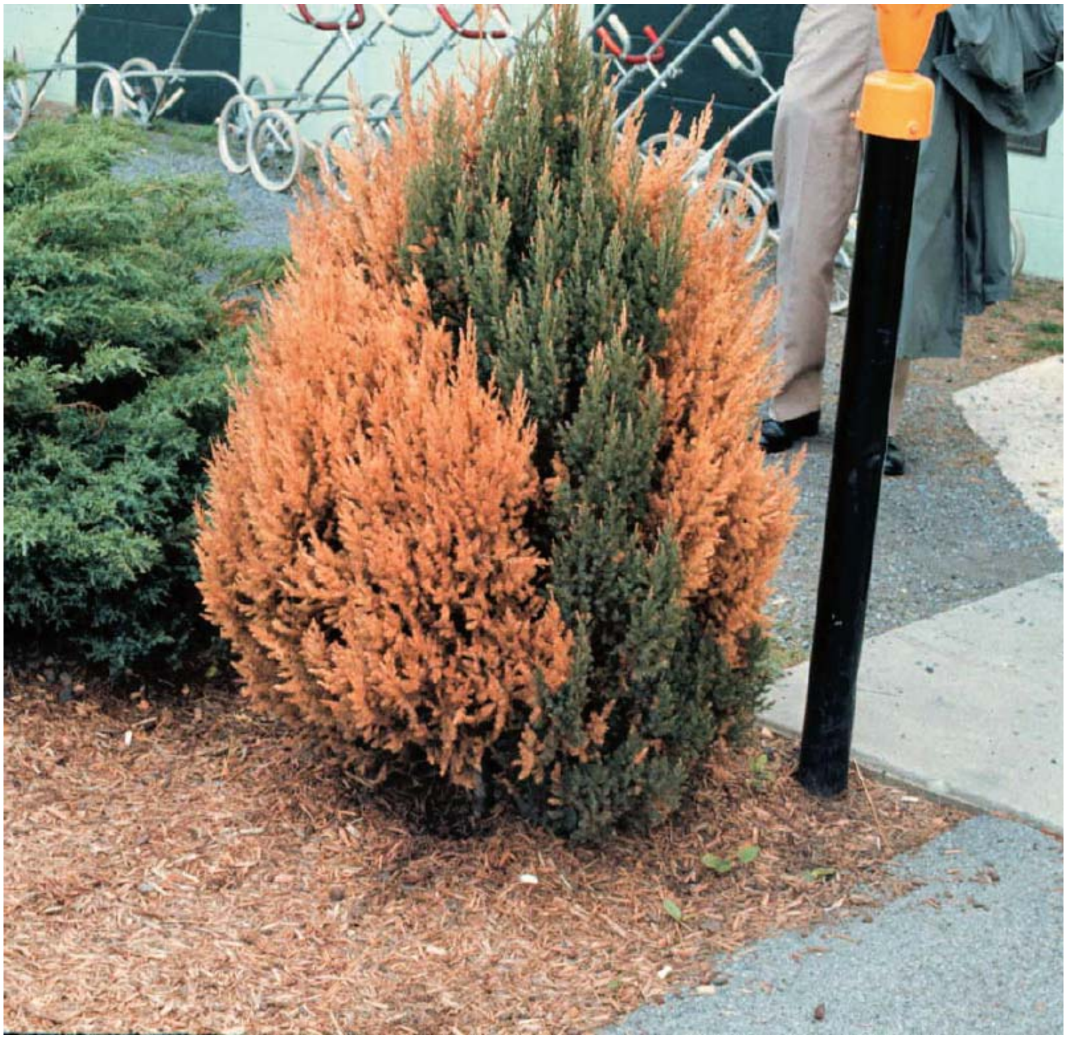

Figure 7.13. Winterburn on juniper.

Plants in flooded fields or poorly drained containers often become pale green or chlorotic and wilted. Continuous flooding can lead to death in days for annual plants and in weeks for trees. Death from excess water is due to root death, caused by lack of the oxygen required for respiration. Excessive soil moisture and high humidity can result in the abiotic disease **edema** (oedema) (Figure 7.12C), in which epidermal cells enlarge because plants have more water than they can use. Edema is common on lower leaf surfaces of overwatered potted plants. The enlarged cells appear blistered and may become corky and coalesce, causing many people to worry that a biotic pathogen is present.

Light and wind often cause relatively obvious damage to plants. Sunscald, which occurs on areas of soft tissue exposed to excess light, usually is first seen as a water-soaked area that later becomes necrotic and often sunken (Figure 7.12E). The symptoms are found only on the side of leaves or fruits facing the sun, which is a good clue in diagnosis. Insufficient light is often a problem with houseplants, especially in the winter, when the plants may become **etiolated** (stems are elongated) and have pale leaves that drop off prematurely. Plants that are moved to areas with lower light levels may drop their lower leaves. Wind damage to plants can be quite dramatic, sometimes resulting in **lodging**. Strong winds during thunderstorms can flatten entire fields of wheat (Figure 7.14), while hurricanes can lodge stately palm trees.

Figure 7.14. Lodging in winter wheat.

Nutrient deficiencies and toxicities produce a wide range of symptoms in plants, depending on the plant species, on how far the nutrient concentration is outside the normal range, and on the environmental conditions (Figures 7.2B, 7.4, and 7.15A–C). However, symptoms in different plants caused by the deficiency of a particular nutrient often are similar because the nutrient has a similar function in many kinds of plants. The most common nutrient deficiencies are for N, P, and K. Chlorophyll production is reduced in N-deficient plants, which often are stunted, with pale green or even yellow older leaves. Plants with insufficient P also grow poorly, and leaves may display red, purple, or brown discoloration. The red or purple color also may be observed in cold weather when plants develop temporary P deficiency symptoms due to reduced root function rather than to a true deficiency of P in the soil. Leaves of K-deficient plants often become chlorotic at the tips or edges, which sometimes then become necrotic. Micronutrient toxicity diseases are most often caused by excesses of B, Mn, and Cu. B toxicity can cause leaf chlorosis and necrosis, whereas toxic levels of Mn can cause leaves to crinkle. Cu toxicity can cause chlorosis and reduced root and shoot growth.

► DID YOU KNOW?

Several different potassium compounds are commonly referred to as “potash.” Originally, the ashes from burned trees were boiled in pots. The liquid eventually contained potassium hydroxide (lye), which was used with fats to make soap. Most of the other compounds sank to the bottom of the pot, forming a black sludge, which was transferred to another pot and boiled until dry. This residue was called “pot-ash.” It contained high concentrations of potassium and was used to fertilize fields in colonial America.

Figure 7.15. Symptoms caused by chemicals. A, Nitrogen deficiency in rice (left). B, Iron deficiency in bluegrass. C, Boron toxicity in highbush blueberry. D, Ozone flecking on tobacco (left). E, Acute sulfur dioxide injury on soybean. F, Epinasty of sugar beet caused by the herbicide picloram. G, Salt toxicity in turfgrass.

Some nutrients are mobile in the plant and move from older tissue into new tissue when amounts are limited. For example, the new growth of an N-deficient plant may look normal, but the older leaves exhibit the yellowing symptom. Other nutrients are not mobile, so deficiency symptoms appear first in the newest growth. Fe, Cu, Mn, and Zn deficiencies may cause chlorosis of younger tissue.

Dog spot is a common abiotic problem in home lawns. Dog urine rapidly desiccates turfgrass leaves, causing a roughly circular spot of dead leaf blades. The release of nitrogen from the urine can stimulate grass growth around the edge of the dead spot. This is more obvious in low-fertility lawns. The size of the spot depends on the size of the dog. The same symptoms are associated with fertilizer spills.

Symptoms Due to Chemical Injury

Air pollutants also cause a range of symptoms in plants (Figures 7.6, 7.8, 7.9, and 7.15D and E). Factors that influence symptom expression include the plant species, the concentration of the pollutant, and the length of exposure. Symptoms of ozone damage can include chlorosis and necrosis that resemble normal senescence. At high concentrations, ozone exposure can result in severe necrosis. At lower exposures, tissues may senesce earlier than normal. Affected tissue also may bronze (turn golden brown) or redden. More diagnostic symptoms of ozone damage include necrotic **flecking** on the upper leaf surface of broadleaf plants (Figure 7.15D) and needle **stippling** on conifers. Ozone enters the leaves of plants primarily through stomata, where it dissolves in the fluid at the cell surface and moves into the cell through the cell wall and membrane. Secondary products generated during this process are thought to cause the symptoms. In contrast, symptoms caused by PAN are more common on lower leaf surfaces, where the nitrates accumulate in the spongy parenchyma and sometimes create a "silver leaf" symptom, as well as overall chlorosis and necrosis.

► **DID YOU KNOW?**

Many spinach cultivars are sensitive to ozone. Spotting develops on exposed leaves, making them unmarketable. Acute ozone injury routinely occurs on Long Island, New York, and has almost eliminated the crop from that region.

► **DID YOU KNOW?**

The ozone layer in the upper atmosphere protects us from ultraviolet radiation. Without it, life on land would be impossible. A number of modern chemicals, such as the chlorofluorocarbons (CFCs) used for refrigerants, solvents, and foam blowing agents, threaten this protective layer in the stratosphere. In contrast, ground-level ozone is a pollutant that harms plants, animals, and humans and plays a role in global warming.

SO_2 and NO_x at low concentrations typically cause interveinal yellowing of leaf tissue and stunting of growth (Figure 7.15E). At higher concentrations, plant tissues may actually bronze or bleach (turn white). Acute exposure can cause interveinal necrosis on broadleaf plants and tip necrosis on conifers. Ethylene, a natural plant growth regulator, hastens aging and senescence in exposed plants. Symptoms caused by exposure to this pollutant include reduced or abnormal growth, premature leaf or petal drop, and cupping of leaves.

► **DID YOU KNOW?**

The effects of sulfur dioxide (SO_2) on plants have been studied for centuries. In 1871, it was the first air pollutant to be designated as phytotoxic.

The adverse effects of acid rain on the populations of microorganisms, insects, and fishes in streams and lakes are well documented. Adverse effects on forest trees also have been demonstrated, including chlorosis, defoliation, and stunting (Figure 7.8). Some studies of field crops have failed to demonstrate harmful effects of acid rain, probably due to the buffering capacity of the soil. For example, when numerous corn and soybean cultivars were watered with rain as acidic as pH 4.3 for an entire growing season, no symptoms and no reproducible yield reductions were found.

Herbicides cause a variety of symptoms in plants, including stunting, distortion, and defoliation of entire plants, as well as leaf distortion, chlorosis, and necrosis (Figure 7.15F). Some symptoms caused by herbicides can be confused with those caused by viruses (e.g., mottles) or by fastidious prokaryotes (e.g., malformations). Horticultural oils and some other pest-control products applied in hot weather can cause browning of foliar tissues.

Direct application of salt to roads to melt winter ice and snow can burn foliage of roadside plants, causing scorch, tipburn, and necrosis (Figure 7.15G). High concentrations of salts in the soil can create drought conditions by interfering with the normal absorption of water by plant roots. Plants may wilt and defoliate when salt concentrations in the soil are high.

How are abiotic diseases diagnosed?

Abiotic diseases are often difficult to diagnose because many factors can cause similar symptoms, and these may be similar to those induced by various biotic pathogens (e.g., Figures 7.11 and 7.12D). For example, it may be difficult to distinguish between short-term drought effects and chronic effects of nutrient deficiencies and air pollutants because the symptoms can be similar. However, accurate diagnosis is very important, because one-third to one-half of the samples that come into plant disease diagnostic clinics have problems caused by abiotic factors, and some of these problems can be managed. The difficulty of diagnosis is increased by the fact that the abiotic factor responsible for the disease may not be present in the plant sample.

A wide range of disease symptoms is induced by abiotic factors, many of which resemble those caused by biotic pathogens. Sometimes no obvious symptoms are present. Other times, symptoms are confounded by the presence of more than one abiotic or biotic cause.

Challenges in the Diagnosis of Abiotic Diseases in Plants

- Many abiotic factors induce similar symptoms.
- Symptoms caused by abiotic factors can resemble those caused by biotic pathogens.
- The causal abiotic factor is often not present in plant tissue, or even in the surrounding environment, at the time of diagnosis.
- Secondary biotic agents may invade tissue weakened by abiotic diseases.

In addition, plants weakened or injured by abiotic factors are often more susceptible to attack by biotic pathogens (Figure 7.16). For example, *Botrytis* infection is greater in plants with necrotic foliar flecks caused by ozone injury or with yellowed lower leaves resulting from N deficiency, low light, or crowded growing conditions. In contrast, rust and powdery mildew, caused by biotrophs that require living cells, may actually be reduced in plants with leaf necrosis and chlorosis. Fire blight and many foliar bacterial diseases of flower and vegetable crops are more common after thunderstorms, and many canker and wood decay pathogens enter plants through wounds created by storms and wind. In addition, various environmental stresses, such as drought, nutrient deficiency, and air pollution, increase the susceptibility of woody plants by impairing their natural defense responses against biotic pathogens.

Figure 7.16. A sugar maple stressed by parking lot paving (left) has developed Armillaria root disease.

Several clues may lead a diagnostician to suspect that an abiotic factor is responsible for a plant disease. First, no signs of a biotic pathogen are present on the diseased samples. Of course, the presence of secondary invaders and saprophytes may cause confusion. Second, symptoms may be at about the same stage of development. In contrast, plants with a disease caused by a biotic pathogen are more likely to display different stages of symptom development, often including both older lesions and younger ones. However, episodic exposures to a harmful abiotic factor can cause lesions of different ages, which can be confusing. Third, abiotic lesions tend to have sharp borders between healthy and affected tissue rather than the yellowish margin of dying cells in the transition from healthy to necrotic tissue in fungal and bacterial lesions. Scorch and tip blights are common abiotic foliar symptoms.

Clues That a Disease Is Abiotic

- Symptoms appear suddenly.
- Symptoms on individual plants are fairly uniform.

- Symptoms develop on several parts of individual plants or on many plants.
- Different plant species in the area have similar symptoms.
- Symptoms may follow a chemical application pattern in lines or rows.
- Foliar lesions have a distinct line between healthy and affected tissue.
- Biotic signs are not present (unless the affected plant has been invaded by secondary pathogens or saprophytes).

In the field, plants with abiotic symptoms may be uniformly distributed rather than in the patchy pattern of diseased plants commonly seen with biotic problems. Affected plants may follow an application pattern in a field or landscape if an herbicide, fertilizer, or other agricultural chemical is misapplied. Symptoms may occur in strips if there have been overlaps or skips, or they may follow the pattern of a chemical spill (Figure 7.17). An abiotic factor may affect many different plant species in the same area, whereas biotic pathogens are often more host specific. If both the weeds and the desired plants in an area exhibit similar symptoms, an abiotic factor is a likely suspect.

Figure 7.17. Phytotoxicity from fungicide spilled on turfgrass.

It is general practice to rule out a biotic cause for a disease before proceeding to explore abiotic causes. One reason for this is that a lot of information must be gathered and assessed to determine the specific abiotic factor responsible for the disease. First, the type, location, pattern, and sequence of development of symptoms on the plants should be noted, as well as the pattern of affected plants in the greenhouse, field, or forest.

Questions about cultural practices may lead to a diagnosis of a nutrient or herbicide problem, which may be confirmed by careful examination of plant tissue for evidence of damage associated with herbicide residue or by analysis of the plant tissue and/or surrounding soil. It is common to submit soil samples from potted plants for analysis of nutrient status and accumulation of salts. Foliar nutrient analysis may be done for plants that are not growing well and have no apparent biotic disease. These tests may even be done routinely for highly maintained turfgrass and certain valuable perennial ornamentals. Identification of nearby sources of air pollution can confirm a diagnosis of NO_x, O_3, or SO_2 damage. Ethylene injury is most common in greenhouses with improperly vented gas or oil heaters.

Checklist of Diagnostic Questions

Affected plants

- What types of plants (and cultivars) are affected?
- What are the ages and sizes of affected plants?
- Where were the plants grown, and where were they purchased?
- Are other nearby plants affected?
- How closely related are the affected plants?

Symptoms

- What plant part(s) is (are) affected?
- What are the symptoms?
- Are any signs of a pathogen present?
- What is the pattern of symptoms on individual plants?
- What is the pattern of disease in the field or area?
- When were symptoms first noticed?
- Are symptoms getting worse or staying the same as when they were first observed?

Environment

- Is the plant species suited to the location?
- Could the planting site be a factor?
- Could soil be a factor?
- Could weather be a factor?
- What are the recent weather patterns?
- What have been the weather patterns for the last few years?

Cultural

- Could the planting method be a factor?
- Were any pesticides used in the area?
- What watering and fertilization practices were used?
- Did the grower apply any treatment for the problem?
- Did the treatment have any effect?

Other

- Has the grower had previous problems of any kind with this type of plant?
- What does the grower think might have caused the problem?

Questions about recent and historical weather patterns may lead to a diagnosis of the environmental factor responsible for the problem. Rainfall and temperature patterns may be quite localized, so diagnosticians should be provided with specific information about the weather preceding the appearance of plant symptoms. Symptoms on trees and other woody plants may not be evident until a year or two after a severe drought or flood, so long-term weather patterns may need to be examined.

Diagnosis of abiotic diseases usually first involves elimination of possible biotic agents. Information about the type, location, pattern, and sequence of appearance of

symptoms, as well as about cultural practices and weather patterns, can be useful.

After all of this sleuthing, possible and even probable causes may become evident. The next step is to consult the literature to look for symptom descriptions that match those found in your situation. Although some symptoms caused by abiotic factors are quite general, on a given plant species they may be quite diagnostic. However, nutrient deficiency or toxicity symptoms found in plant diagnostic guides may be misleading, because the symptoms are sometimes produced in a laboratory by withholding a single nutrient or by adding an excessive amount of one in a hydroponic solution. In the real world, it is more likely that more than one nutrient is deficient or toxic in the same soil.

To conclusively prove that an abiotic factor is the cause of a particular disease, a scientist must reproduce the disease on healthy plants by subjecting them to the same conditions that created the original problem. This is often not a simple task, especially for perennial plants. It is much easier to recreate acute effects than chronic ones.

How are abiotic diseases managed?

Some abiotic diseases cannot be managed. Plants are sedentary and cannot escape local changes in their environment, such as natural droughts or floods. However, other abiotic diseases are caused by human cultural practices or other activities and can be effectively managed. Approaches to management also must consider whether there is a risk that a biotic pathogen is present that can multiply and spread to new host plants.

Although plants cannot escape environmental stresses, diseases caused by these abiotic factors can be minimized by growing plants that are best adapted to the local environment. Cold damage can sometimes be ameliorated by providing additional heat, spraying plants with water when freezing temperatures are expected, or using wind to mix warmer air with the cold air. High value plants also can be protected from cold with mulches, wraps, or cold frames. People can change the plant's local environment in other ways, such as by installing drainage systems that reduce flooding or by planting windbreaks that lessen the effect of prevailing winds. Scientists are exploring how we can take advantage of plants' natural mechanisms to develop crops that can survive in colder, more arid, or more saline conditions.

► **DID YOU KNOW?**

Some epiphytic bacteria serve as nuclei for the formation of ice on plant surfaces. These bacteria are produced commercially in large quantities for use in snowmaking at ski resorts. When the bacteria are mixed in water, more snow is produced per liter (gallon) of water. Some of these bacteria have been genetically engineered so that they can no longer cause the formation of ice. These "ice-minus" bacteria can be sprayed on sensitive plants, such as strawberries, to protect them from frost damage.

Sufficient water is critical to good plant health, and supplemental water should be applied on a regular basis when rainfall is not sufficient. Blossom-end rot disease of tomato and pepper is caused by a Ca deficiency that results when the water supply is irregular (Figure 7.18). Winterburn occurs when soils are frozen, and water is unavailable to plants. The condition is exacerbated by dry wind blowing across plants that keep their leaves in the winter, such as azaleas, rhododendrons, and various conifers. Planting sites in open areas where wind routinely blows in the winter should be avoided. Landscape plants are sometimes sprayed with commercial formulations of waxes or polymers to help reduce water loss from the leaves.

Figure 7.18. Blossom-end rot of tomato caused by calcium deficiency.

Supplemental light often must be provided for plants growing in buildings and greenhouses in the winter. In contrast, young plants accustomed to the less intense light of a greenhouse should be introduced to full sun gradually and planted outdoors on cloudy days.

Most macronutrients are limited in soil, so it is common to apply these elements to soils in production fields, greenhouses, home gardens, and pots. Nutrients can be applied in various forms, such as manure, **green manure** (a crop plowed under while still green), composts, and chemical fertilizers. N, P, and K are needed in the greatest quantities by agricultural plants and therefore are the most common components of commercial fertilizers. However, before fertilizer is added to a soil, the soil should be tested to determine its pH and which nutrients are required. High concentrations of quickrelease N fertilizers can burn plants, may be leached from the soil before being taken up by plants, and may cause an increase in the population of detrimental fungi and bacteria. Organic amendments are advantageous because N becomes available to the plant gradually as the organic material is degraded, and all of the required nutrients are supplied.

The availability of elements present in soil can be altered by adjusting the soil pH. Soil pH between 6 and 7 is generally considered best for most plants. Lime, which contains Ca, is often applied to increase the pH of field soil. The higher pH increases the availability of some macronutrients (e.g., Ca, K, and Mg) and reduces the availability of all micronutrients (except Mo), thereby decreasing the likelihood of toxicity diseases from elements such as Al and Mn. If soil pH is above 7, Fe is often unavailable. Ammonium nitrate or sulfate, urea, and elemental sulfur are some materials that can be applied to lower the soil pH.

► **DID YOU KNOW?**

Gray speck disease in oats is caused by bacteria that live in the rhizosphere of "susceptible" oat cultivars. The bacteria oxidize manganese, thus making it unavailable to the oat plants, which then develop gray speck symptoms of manganese deficiency in leaves. The bacteria do not multiply well around the roots of resistant oat cultivars, which remain healthy.

► **DID YOU KNOW?**

The numbers 30-30-10 on a fertilizer label mean that when you spread 100 pounds of the fertilizer, you are adding 30 pounds of nitrogen, 30 pounds of phosphorus, and 10 pounds of potassium.

Nutrient availability can be altered by changing the soil moisture or tilling the soil. Irrigation can increase the solubility and thus the availability of many elements. Also, the pH of irrigation water from city water supplies may differ from the soil pH and affect nutrient availability. Tillage exposes soil to air, which stimulates microbial activity that releases many elements from organic matter in the soil. These elements then become available to plants.

Emission standards for air pollutants produced by human activities have tightened over the years, but industrialization and urbanization continue to increase the number of sources of these pollutants. For example, air quality standards for ozone have existed in the United States since 1970, but levels of ozone in the lower atmosphere have not decreased and remain at levels that reduce the yields of major crops. There is genetic variation in ozone sensitivity in plant species (Figure 7.19). Breeding programs have tried to select cultivars of spinach, tobacco, and other species that have greater tolerance to ozone for planting in areas where ozone levels are consistently high. Despite our knowledge that ozone is the most significant air pollutant in terms of damage to both agricultural crops and forest ecosystems, it has not ever been adequately controlled. Even though emission standards for automobiles have become stricter, there are more cars being driven more miles and increased numbers of large, heavy vehicles (e.g., SUVs) that are not required to meet these standards.

Figure 7.19. Genetically based variation in sensitivity to ozone in white pine.

In contrast, the control of SO_2 emissions by power plants in the United States is a remarkable success story. Since laws regulating emissions were first passed in the early 1990s, emissions are down by half, at a fraction of the cost originally estimated. For example, the installation of “scrubbers” on coal-burning power plants eliminates up to 95% of the sulfur emissions. Technology also exists to reduce hydrogen fluoride emissions by industries that produce it. **Indicator plants** (plants that are susceptible to very low levels of a given pollutant) are sometimes used for bioassays to detect specific air pollutants and to determine whether emission reduction technologies are working.

Herbicide damage can be managed by following the directions on the label, by applying these chemicals when wind speed is less than 16 kilometers per hour (10 miles per hour), and by using the appropriate application equipment that has been properly calibrated. “Chemical trespass” of any agricultural chemical, including herbicides, fungicides, insecticides, horticultural oils, and fertilizers, can be avoided by not spraying under windy conditions or when sudden rainstorms are more likely to wash contaminants into surface waters or adjoining soils. It also is wise to avoid applying herbicides and oil-based formulations of other chemicals in hot weather, especially if plants are stressed by drought, because phytotoxicity is more likely to occur. Volatile herbicides can contaminate planting media when both are housed in the same closed storage area. Therefore, they should be stored separately.

In areas where salt is applied to melt ice and snow, salt-contaminated snow should not be piled on turfgrass and low growing ornamentals, such as junipers. In the spring, salt-contaminated areas can be flushed with irrigation. To help avoid salt buildup in the root medium of potted plants, the plants should be watered enough to wash excess salts out through the bottom of the pot.

Human Management of Abiotic Diseases

	Environmental factors	**Chemicals**
easier to manage	nutrient deficiencies light for indoor plants	herbicide application, drift pollution from a point source (e.g.,

		a smelting factory)
harder (sometimes impossible!) to manage	nutrient toxicities freezing temperatures drought	herbicide carryover pollution from a dispersed source (e.g., automobile exhaust)

Some abiotic diseases lend themselves to management, such as elimination of nutrient deficiencies through fertilization and prevention of herbicide misapplication by following label instructions. Other diseases are difficult or impossible to manage, such as freezing temperatures, drought, and air pollution from many sources (e.g., motor vehicles).

REFERENCES

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Questions

1. Define “disease” and “injury” as they relate to plants. Give an example of an abiotic factor that causes each.
2. Survey a landscape area near you for evidence of winter-burn if you live in a climate with a cold winter. What symptoms will you look for? How could you reduce injury in coming years?
3. What is the difference between a macronutrient and a micronutrient? What macronutrients and micronutrients are required by plants? Where do plants get the majority of their nutrients?
4. How does soil pH affect nutrient availability? How can you adjust soil pH?

5. What is the difference between a primary and a secondary air pollutant? Give an example of each.
6. How does diagnosis of abiotic diseases of plants differ from diagnosis of biotic diseases? What clues might lead you to suspect that an abiotic factor is responsible for a disease?
7. Can Koch's postulates be completed for an abiotic factor? Why or why not?
8. A plant exhibits the following symptoms: stunting, wilting, leaf scorch, and yellowing. What possible biotic and abiotic causes of these symptoms will you consider in your diagnosis?
9. What kinds of abiotic problems might be diagnosed by testing the tissues of affected plants or the soil surrounding the plants?
10. Some abiotic diseases can be managed by people, whereas others cannot. Give an example of a disease that can be managed and explain how. Give an example of a disease that cannot be managed and explain why not.
11. Ozone levels exceed environmental standards in much of the United States and cause problems for plants, animals, and people. What are some possible ways to reduce atmospheric levels of ozone?

Challenge Questions

1. Should plant problems caused by abiotic factors be considered diseases or is there another term that is more appropriate? Do some abiotic problems qualify as diseases whereas others do not?
2. Why is it so difficult to predict the impact of changes in environmental factors, such as acid rain, ambient temperature, and atmospheric ozone, on plant health?
3. What methods of fertilizer application to plants can help minimize deficiencies and toxicities and susceptibility to biotic pathogens? How are fertilizers applied to plants of interest to

you? How might you modify these fertilizer applications to improve plant health?

Supplementary Resources

Illustrated Glossary

Special Topic

Plant disease diagnosis

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

abiotic
acid precipitation
acute
air pollution
chronic
drift
drought
edema (oedema)
ethylene
etiolation
fleck
forest decline
green manure
herbicide
hydrogen fluoride (HF)
indicator plant
injury

lodge
macronutrient
micronutrient
nitrogen oxide (NO_x)
ozone (O_3)
peroxyacetyl nitrate (PAN)
pH
photochemical reaction
primary pollutant
scorch
secondary pollutant
stippling
sulfur dioxide (SO_2)
sunscald or sunburn
temporary wilt
winterburn

Exercise

Exercise 7.1. Make a preliminary diagnosis as to whether the problem in each photograph is caused by an abiotic or a biotic disease agent. Explain your reasoning for each choice.

Internet Resources

APS Resource

Non-infectious diseases of common houseplants

Recommended Websites

AIRNow—Quality of Air Means Quality of Life
Effects of Ozone Air Pollution on Plants

How To Identify and Control Noninfectious Diseases of Trees
How To Identify Ozone Injury on Eastern Forest Bioindicator Plants
U.S. Environmental Protection Agency

Internet Research

Internet Research Exercise 7.1. What abiotic diseases are common near your home? Are there particular plant species that you would avoid planting because of these problems?

Internet Research Exercise 7.2. Go to the U.S. Environmental Protection Agency site AIRNow—Quality of Air Means Quality of Life.
Select a region of the United States. Are high levels of atmospheric ozone a problem in this region? You can find archived maps and animations for various states and regions by searching for "ozone map archives." Ozone levels are highest during what months? Why? Ozone levels are highest during what hours of the day? Why?

Internet Research Exercise 7.3. What are the symptoms of ozone toxicity on three plant species of interest to you? Are any ozone-tolerant cultivars of these plants available?

Internet Research Exercise 7.4. Where can you get soil and tissue nutrient analyses done near you? What kind of sample do they require?

CHAPTER 8

What Types of Plant Diseases Are There?

IN THIS CHAPTER:

- **What is damping-off?**
- **What diseases commonly affect foliage, flowers, and fruits?**
- **What are vascular diseases?**
- **What are cankers?**
- **What are galls?**
- **What are root rots?**
- **What is wood decay?**
- **What are postharvest diseases?**

Chapters 2 through 6 describe diseases caused by specific groups of pathogens. In many cases, we are confronted by a diseased plant but do not know what kind of pathogen is responsible. This chapter describes common diseases based on the symptoms they cause in plants. Each section includes some important examples of diseases and describes some of their specific symptoms and signs. These descriptions may suggest a preliminary diagnosis and management practices that may be successful in managing a disease.

It is sometimes possible to identify a disease simply by looking at the plant. For example, powdery mildew is a disease caused by ascomycetes that typically produce a white mass of mycelium and powdery conidia on the surface of infected leaves. These signs can be easily seen, even without a microscope.

Other diseases, such as blights and cankers, can result in significant amounts of **necrosis** (death of tissue). These diseases can be caused by a variety of fungi and sometimes bacteria, so laboratory analysis is usually needed to determine the primary causal agent of a disease. Root rots may not be noticed until the aboveground plant parts begin to exhibit symptoms, after the disease has progressed for some time. Diagnosis may require isolation of a pathogen from the infected roots or completion of a nematode assay. Viruses and certain fastidious bacteria can cause symptoms that are similar or identical to those caused by nutrient deficiencies and other environmental stresses, so a complete history and various laboratory tests may be required to confirm a diagnosis.

Diseases of Foliage, Flowers, and Fruits: Diagnostic Symptoms and Signs

necrotic spot (plus blight, blotch, needle cast, scab)

- round to oval spots (pathogens often fungi)
- angular spots (pathogens often bacteria or foliar nematodes)

anthracnose

- pathogens are ascomycetes
- dark, sunken lesions often observed
- sticky masses of conidia in acervuli, perhaps with setae

powdery mildew

- pathogens are ascomycetes
- white superficial mycelium and powdery conidia
- dark, round chasmothecia (cleistothecia) filled with asci and ascospores

downy mildew

- pathogens are oomycetes
- sporangiophores and sporangia in downy tufts, often on the lower surface of the leaf
- oospores in infected tissue
- necrotic areas on leaves and/or fruit

rust

- pathogens are basidiomycetes
- variously colored spores (urediniospores, teliospores or aeciospores, depending on host), but powdery, orange- to rust-colored urediniospores are seen most often

smut

- pathogens are basidiomycetes
- black, powdery teliospores
- often distorted growth

virus disease

- mosaic, mottle, ringspots, veinbanding, yellowing

What is damping-off?

Damping-off is the infection of seeds and seedlings, leading to death (Figure 8.1). It is distinguished from diseases of mature plants because seedling tissues are still quite soft and immature, which makes them vulnerable to attack. In pre-emergent damping-off, a seed may simply rot or fail to emerge because it has been attacked by a pathogen below ground. In postemergent damping-off, seedlings emerge but are infected, usually at the soil line, by a soilborne pathogen. The infected seedlings topple over and collapse. The area of infection may look necrotic and pinched in. In greenhouse flats, damping-off is often evident as circular patches (or foci) of missing or dead seedlings. Damping-off pathogens are usually capable of attacking a wide variety of plants at the seedling stage. Most of these pathogens survive well as soil saprophytes and should be assumed to be present in nearly all soils.

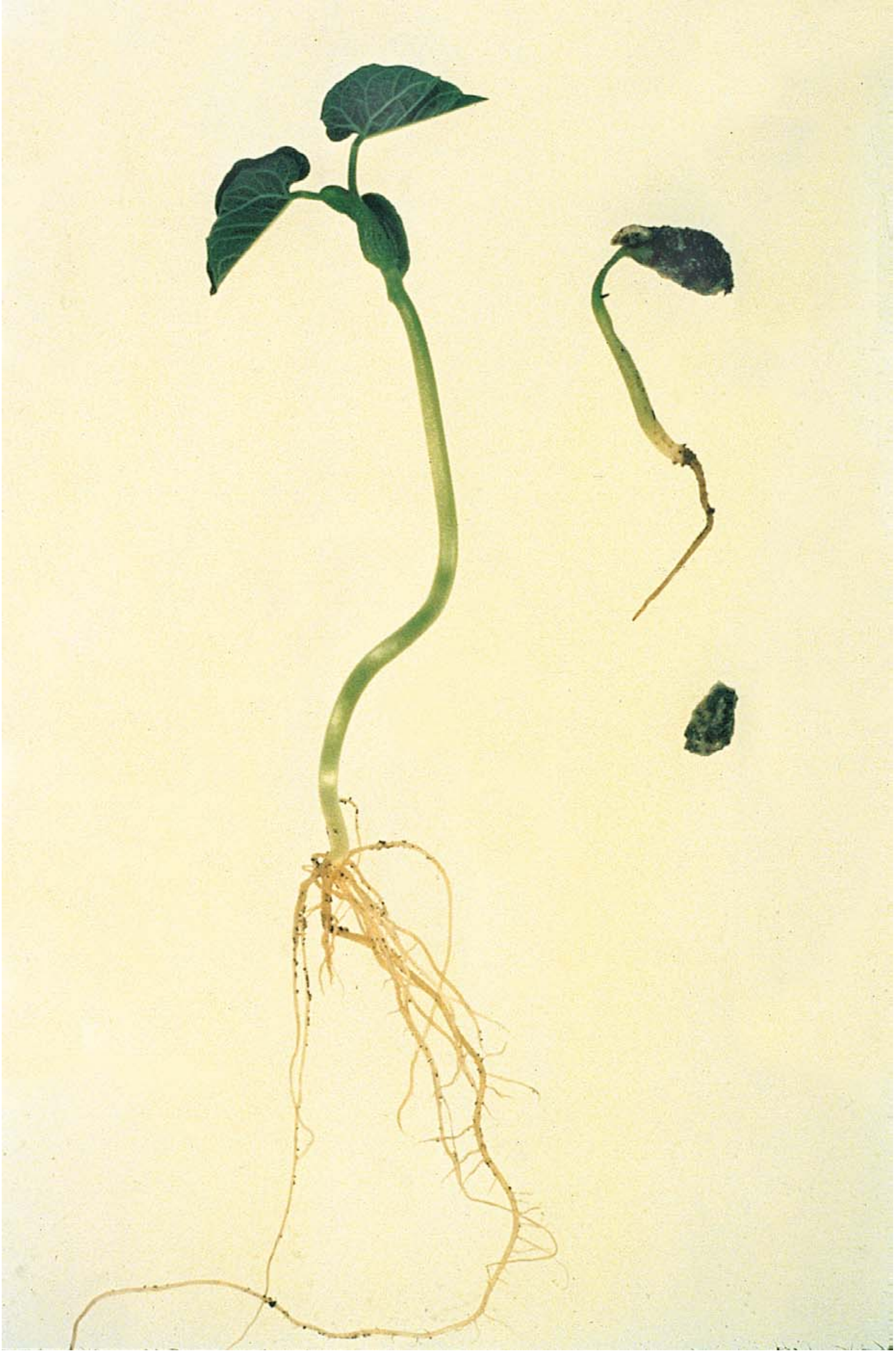


Figure 8.1. Bean seedling affected by damping-off (right) and a healthy seedling (left). Damping-off includes seed rot and pre- and post-emergence seedling death.

Damping-off: infection of seeds and seedlings, leading to death

Damping-off is the infection of seeds or seedlings, leading to death. These diseases are caused by various soilborne pathogens, mostly fungi. Sterile planting media, optimal germination environments, and biological or chemical seed treatments will minimize damping-off.

In excessively wet conditions, *Pythium* is the most common damping-off pathogen. The nutrient-rich exudates of germinating seeds and seedlings attract the **chemotactic** zoospores of *Pythium.* When moisture is not excessive, *Fusarium, Rhizoctonia*, and other fungi are common causes of damping-off. Chemical management is usually not successful once damping-off is observed because the seedlings are too badly damaged to recover. It is usually most cost-effective to reseed damaged areas if the underlying conditions that led to disease can be ameliorated.

The most effective approach to management of damping-off in outdoor soils is **avoidance**. Seeds should be planted in optimal environmental conditions for germination and growth to minimize the time that seedlings are vulnerable to damping-off pathogens. Pathogen-free soil or planting media should be used in greenhouses and for transplants. Biological and chemical seed treatments are sometimes used to protect seedlings during this vulnerable time.

What diseases commonly affect foliage, flowers, and fruits?

Necrotic Symptoms

Numerous pathogens cause foliar necrosis, including limited-area **leaf spots** or expanding areas of dying tissue known as **blotch** (Figure 8.2A). When large areas have been quickly killed, the disease is described as a **blight**. Conifers and other plants with needlelike leaves are susceptible to loss of needles due to foliar diseases called **needlecasts** (Figure 8.2B). Crusty, superficial foliar and fruit lesions are **scabs**. The severity of these symptoms depends on the natural resistance of the plant to the specific pathogen, the virulence of the pathogen, and the environmental conditions.

DISEASE CLASSIC

Bacterial Spot of Pepper and Tomato

Hosts pepper, tomato

Pathogens *Xanthomonas axonopodis* pathovar *vesicatoria* (syn. *X. campestris* pv. *vesicatoria*), *X. vesicatoria, X. gardneri*

Symptoms and Signs Small, brown, angular spots on leaves and fruits, sometimes with a yellow halo. Defoliation when infection is severe.

Disease Cycle Bacteria are commonly introduced with infested seed or transplants. Splashing water and overhead irrigation increase the spread of bacteria. Bacteria enter plants through stomata or wounds. They do not survive for long periods in soil or debris.

Management Growers should use pathogen-free seed and transplants. Some cultivars have genetic resistance. Rotation with nonsusceptible crops reduces disease. Copper sprays can protect plants.

Significance Bacterial spot is a destructive disease in warm, humid climates worldwide.

Plant Disease Profiles: Bacterial spot of pepper and tomato

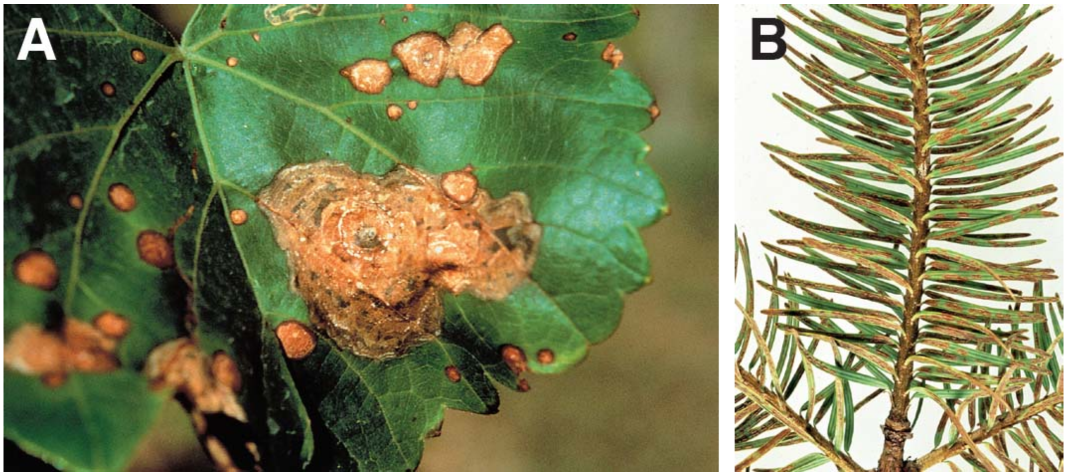


Figure 8.2. Examples of necrotic foliar symptoms. A, Leaf blotch. B, Needlecast; infected needles are eventually cast from the tree (i.e., they fall off).

In general, leaf moisture increases the occurrence and severity of foliar diseases caused by fungi, bacteria, and foliar nematodes. Water favors germination of fungal spores and multiplication of bacteria. Humid conditions also favor spore production on infected leaf surfaces. Foliar nematodes move up stems or leaves from the soil in water films.

Some pathogens are more likely to infect the softer tissues of lush, well- fertilized leaves, causing **high-nitrogen diseases** (Figure 8.3). Others prefer yellowing, senescent tissue, causing **low-nitrogen diseases**. For example, excessive use of spring fertilizers on lawns for the fast “green-up” desired by many homeowners favors the fungi that cause common leaf spot diseases of Kentucky bluegrass and other lawn grasses. The leaf spots can progress into **melting-out** (turf collapse) as leaf spots accumulate and the infection moves into the crown of the plant. In contrast, gray mold caused by *Botrytis* *cinerea* is often a low-nitrogen disease of many annual plants and small fruits, such as strawberry and brambles (Figure 8.4). If plants are nitrogen-deficient or overcrowded, the yellow, lower leaves are more susceptible to infection by *B. cinerea*.

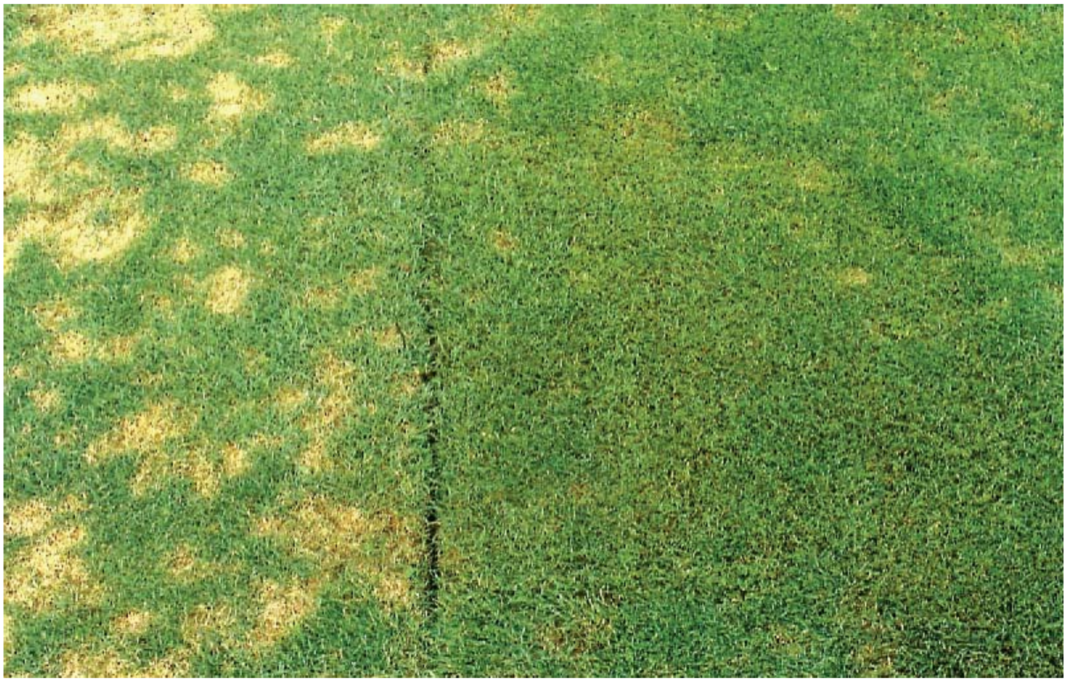


Figure 8.3. Nitrogen levels can affect susceptibility to disease. Brown patch (right) occurs in turfgrass in places where high levels of nitrogen fertilizer were applied. Dollar spot (left) occurs where nitrogen is deficient.

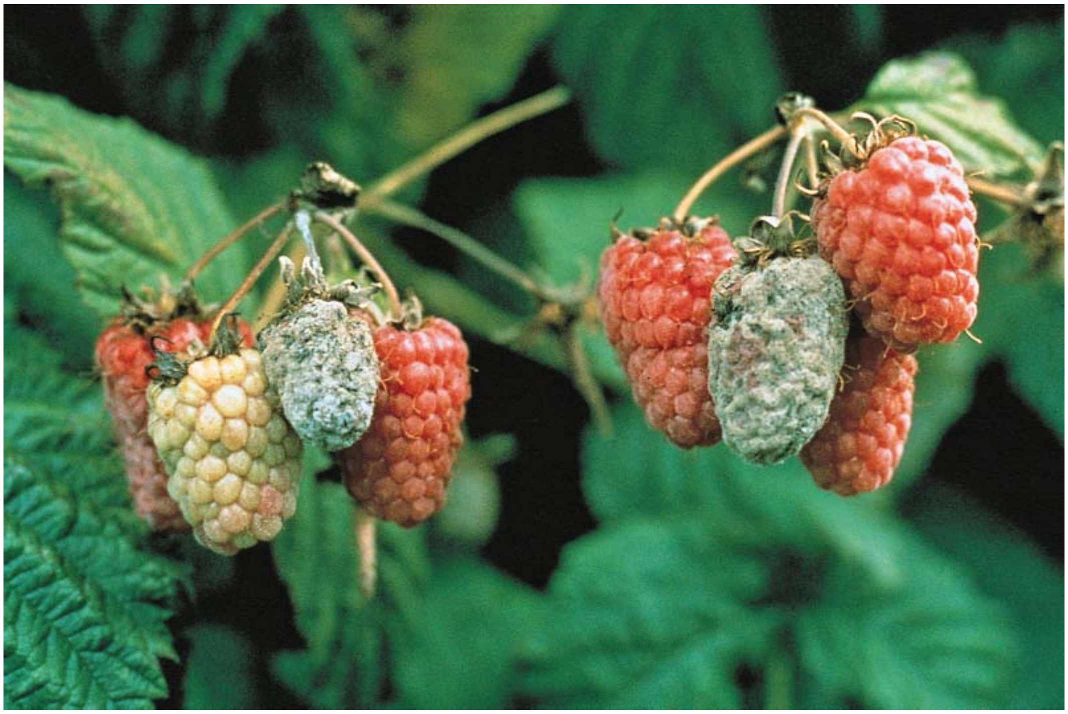
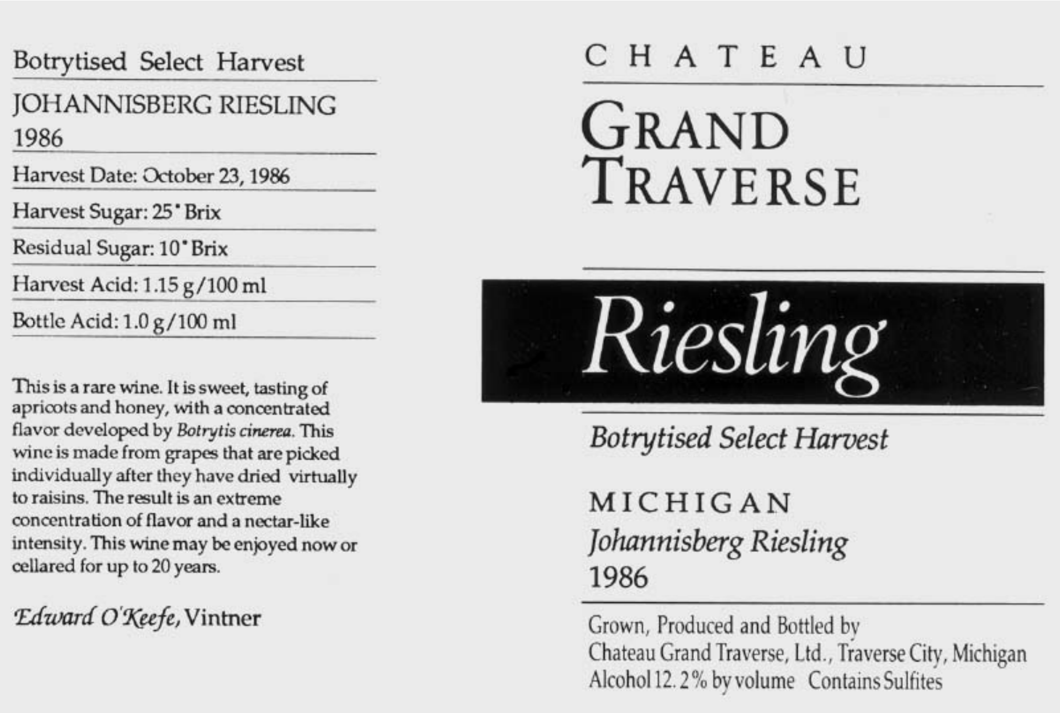


Figure 8.4. Gray mold, caused by *Botrytis cinerea*, on red raspberry fruit. Note the dusty covering of gray spores on infected fruit.

► DID YOU KNOW?

Botrytis cinerea is a common fungal pathogen of many plants. It causes gray mold, the most important disease of greenhouse crops. It also causes bunch rot of grapes. Under the right environmental conditions, *B. cinerea* causes a slow rotting that concentrates the sugars in grapes and gives them a fungal flavor that vintners have found appealing. Gray mold in this form is called "the noble rot," and late-harvest grapes infected under these conditions are used to produce premium wines labeled *botrytised* (Figure 8.5).



Botrytised Select Harvest

JOHANNISBERG RIESLING
1986

Harvest Date: October 23, 1986

Harvest Sugar: 25° Brix

Residual Sugar: 10° Brix

Harvest Acid: 1.15 g/100 ml

Bottle Acid: 1.0 g/100 ml

This is a rare wine. It is sweet, tasting of apricots and honey, with a concentrated flavor developed by *Botrytis cinerea*. This wine is made from grapes that are picked individually after they have dried virtually to raisins. The result is an extreme concentration of flavor and a nectar-like intensity. This wine may be enjoyed now or cellared for up to 20 years.

Edward O'Keefe, Vintner

CHATEAU
GRAND
TRAVERSE

Riesling

Botrytised Select Harvest

MICHIGAN
Johannisberg Riesling
1986

Grown, Produced and Bottled by
Chateau Grand Traverse, Ltd., Traverse City, Michigan
Alcohol 12.2% by volume Contains Sulfites

Figure 8.5. Label of a wine produced from grapes that were "botrytised," or infected by *Botrytis cinerea,* the fungus causing "the noble rot."

Round to oval leaf spots (Figures 8.2 and 8.6) are usually caused by fungal pathogens. The parallel leaf veins of monocots, such as those of lily or iris, can cause fungal leaf spots to become elongated between the veins. Bacterial pathogens or nematodes typically cause **angular leaf spots** (Figures 3.9 and 4.12) because leaf veins stop the pathogens, at least temporarily, as they progress through leaf tissue.

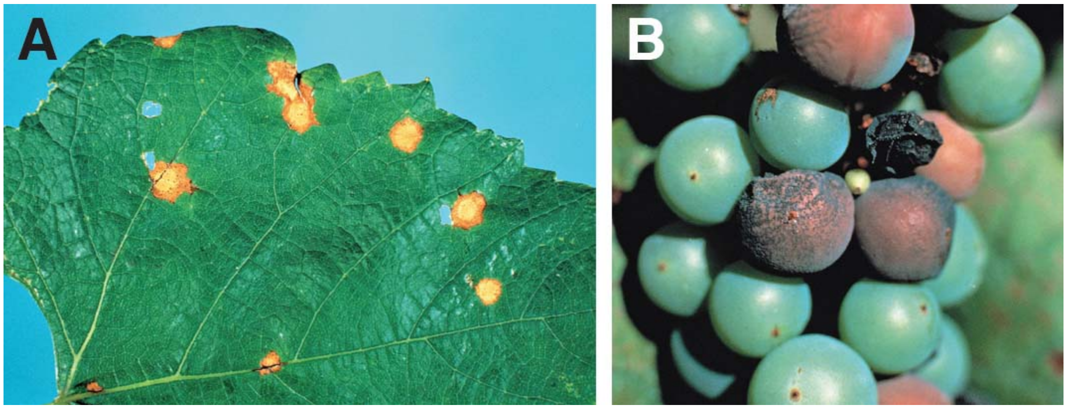


Figure 8.6. A, Black rot lesions on a grape leaf. B, Black rot of a cluster of grapes at various stages of disease.

Diagnosis of foliar pathogens requires observation of signs of the pathogen such as mycelia, spores, fruiting bodies, bacterial streaming, or nematodes. Incubation of infected leaves in high relative humidity (a moist chamber) for 24–48 hours can enhance observation of fungal pathogen signs because many fungi will produce mycelia and spores on the surface of infected tissues. Presumptive diagnosis of bacterial diseases can be accomplished by cutting infected tissue in water on a microscope slide and microscopically observing masses of bacteria exuding into the water (**bacterial streaming**), but the lack of streaming doesn't exclude a bacterial pathogen. Laboratory isolation of some fungal pathogens and all bacterial pathogens is necessary for an accurate diagnosis because many saprophytes and secondary invaders are present in necrotic tissues. Diagnosis is most likely to be accurate in the early stages of disease, when the primary pathogen is most prevalent.

Flowers and fruits are often susceptible to the same pathogens that cause foliar diseases, but the extent of the infection may vary. In **black rot** of grape (Figure 8.6), the fungal pathogen *Guignardia bidwellii* causes limited leaf spots, but it can destroy whole clusters of grapes. The fungus *Botryosphaeria obtusa* causes only the minor “frog-eye” spot on apple leaves, but it causes destructive black rot of apple fruit. This pathogen can even kill the tree as a canker pathogen (see below). *Botrytis cinerea*, which causes **gray mold**, commonly attacks very young tissue, in which case it is a damping-off pathogen, but *B. cinerea* also infects aging flowers. In ornamental plantings and greenhouses, old flowers and other dying tissue should be removed. Plants should be appropriately spaced so lower leaves receive light and dry quickly, which helps reduce disease development. *B. cinerea* infection is an important cause of fruit loss both in the field and after harvest.

Necrotic Foliar Diseases

Plant Disease Profiles:

- **Apple scab** (see Chapter 1, Disease Classic)
- **Bacterial spot of pepper and tomato** (see Chapter 8, Disease Classic)
- **Fire blight of apple and pear** (see Chapter 3, Disease Classic)
- **Late blight of potato** (see Chapter 1, Disease Classic)

Additional disease profiles are provided in Supplementary Resources.

Common necrotic foliar symptoms are leaf spot, blotch, blight, needle cast, and scab. Fungi or bacteria usually cause the diseases with these symptoms. The same pathogens may attack flowers and fruits. Moisture favors these pathogens. Many foliar diseases are considered high-nitrogen or low-nitrogen problems.

Anthracnose

Anthracnose diseases affect many kinds of plants. They are caused by certain ascomycetes that produce **acervuli** (asexual fruiting bodies) filled with sticky **conidia** (Figure 8.7). The acervuli erupt through the epidermis of leaves, herbaceous stems, and fruits and cause significant necrosis in the underlying tissue. Symptoms vary depending on the kind of plant and part of the plant that is diseased. The name *anthracnose* is derived from the Greek word for coal, which describes the black acervuli of the causal fungi. These fruiting bodies may include **setae**, which are whiskerlike fungal structures, usually black, that are also signs of anthracnose. The acervuli are sometimes produced in concentric rings that may be observed on infected fruits and vegetables (Figures 2.2 and 8.8).

DISEASE CLASSIC

Anthracnose of Turfgrass

Hosts cool-season turfgrasses, especially creeping bentgrass and annual bluegrass in golf putting greens

Pathogen *Colletotrichum graminicola* (an ascomycete)

Symptoms and Signs Irregular areas of yellowing turf, especially around the outer edge of putting greens and in poorly drained and shady areas; acervuli with black setae visible on leaves, with a hand lens. Anthracnose may progress until the crowns of infected plants are blackened (crown rot stage).

Disease Cycle Sticky conidia are spread by mowers to wounded plants. The pathogen survives in soil and thatch.

Management Fungicides are effective in the early stages of anthracnose, but not in the crown rot stage. The fungicides reduce fungal growth, but recovery usually requires relief of the stresses that increased the susceptibility of the grass: soil compaction, poor drainage, frequent and low mowing, and insufficient nitrogen. The conidia should be washed from mower blades when the mower is moved from a diseased area.

Significance Anthracnose of turfgrass has become very severe where increased numbers of golfers, longer golfing seasons, and demands for faster putting green speed have increased stress and susceptibility to the disease. Anthracnose is rarely a concern in lawns and other areas where the grass is cut higher.

Plant Disease Profiles: Anthracnose of turfgrass

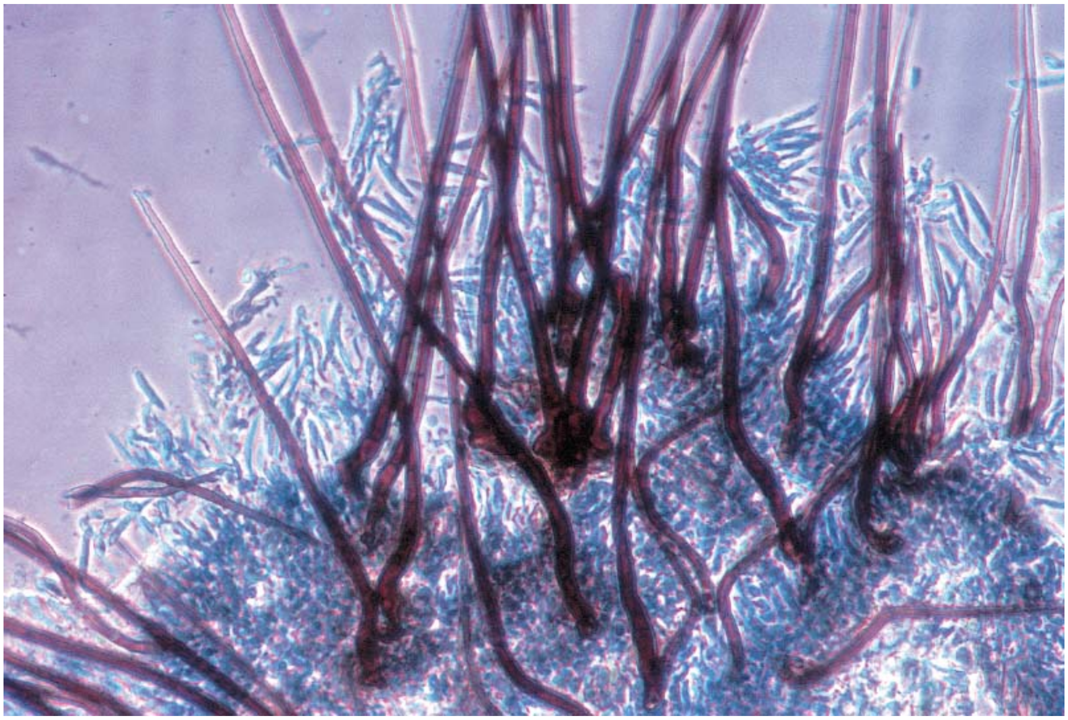


Figure 8.7. Acervulus of an anthracnose fungus observed with a compound microscope. Note the black setae (hairlike projections) and the light-colored conidia. (x400)

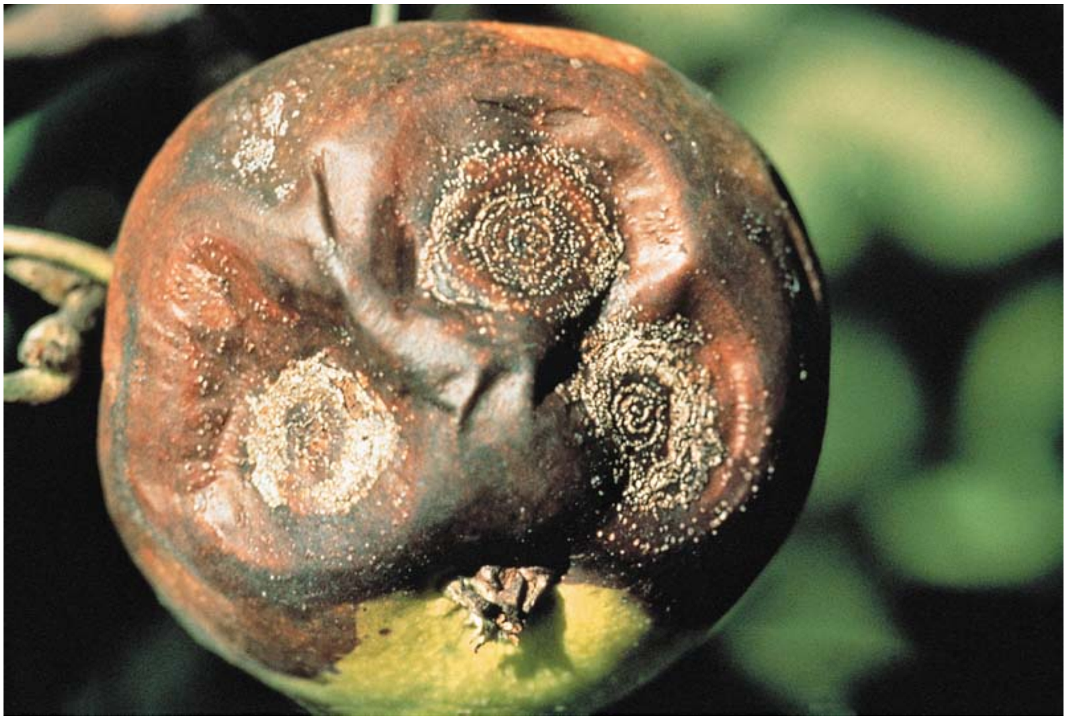


Figure 8.8. Bitter rot, an anthracnose disease of apple, caused by *Colletotrichum gloeosporioides* and *C. acutatum.* Note the concentric rings of acervuli.

Anthracnose diseases are common in humid climates on herbaceous plants, such as beans, curcurbits, strawberry, and cool-season turfgrasses. In woody plants, anthracnose diseases, such as dogwood anthracnose and sycamore anthracnose, begin in the leaves but may progress to form cankers in woody tissues (see below). Because the conidia are sticky, anthracnose pathogens are dispersed by water, insects, human activities, and machinery. Anthracnose diseases are commonly managed by minimizing leaf wetness and removing infected plants and infested plant debris when possible.

► DID YOU KNOW?

Anthrax, caused by the bacterium *Bacillus anthracis*, is a disease of animals. Anthracnose, caused by various fungi, is a disease of plants. The names *anthrax* and *anthracnose* are both derived from the Greek word for coal. In humans, the skin form of anthrax results in blackened tissues. Blackened tissues are also commonly observed in plants with anthracnose diseases.

Anthracnose refers to a group of diseases with variable symptoms, but the common sign is acervuli filled with sticky conidia. The acervuli may include black setae and may be produced in concentric rings. In woody plants, anthracnose may lead to cankers. Wet conditions favor these diseases.

Leaf Blisters and Curls

Species in the ascomycete genus *Taphrina* cause puckering and deformations of leaves. These are symptoms of diseases such as peach leaf curl, plum pocket, and oak leaf blister (Figure 8.9). Naked **asci** (i.e., asci without a fruiting body) are produced on the surface of infected leaves. Because most infections occur on newly expanding leaves, a single early-season fungicide application is usually sufficient to protect orchard trees, where disease may be severe. In ornamental plants, these diseases are usually minor and require no management.

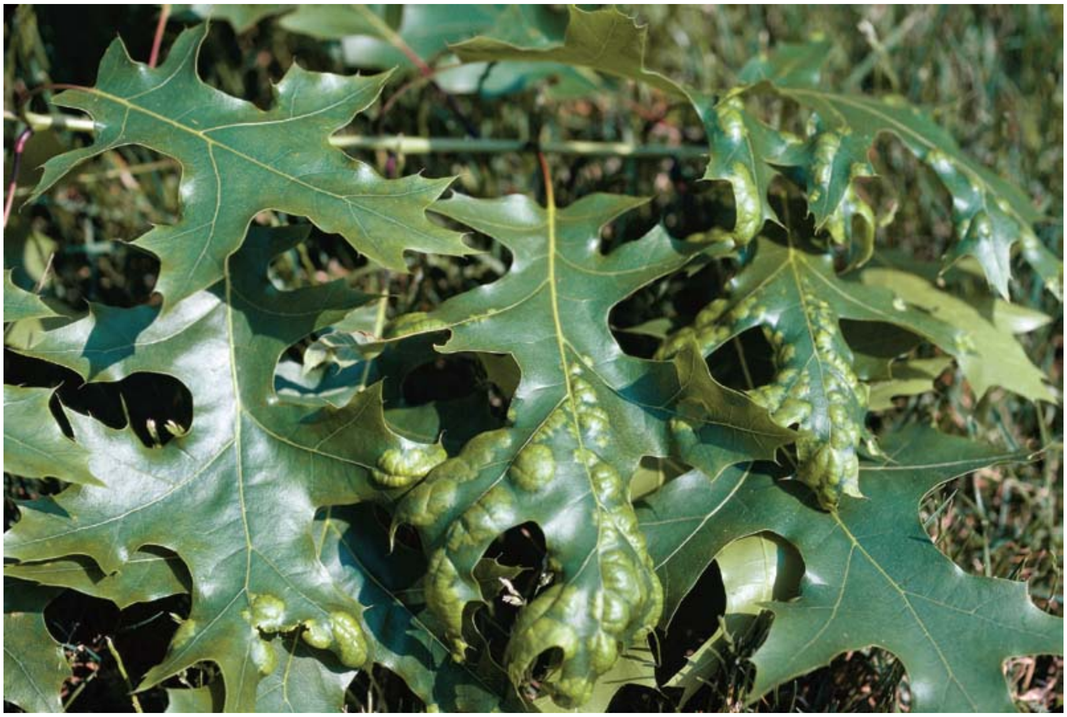


Figure 8.9. Oak leaf blister, caused by *Taphrina caerulescens*.

***Taphrina* spp. are ascomycetes that produce naked asci on leaves. The infections cause leaves to pucker and curl. Fungicides may be required for disease management in orchards.**

Abnormal Color or Form

Foliar symptoms of virus-infected plants include **mosaic**, **mottle**, **ringspot**, **rugosity**, and **vein banding** (Figure 8.10). Symptoms also may occur on fruits and flowers (**flower break**). The viruses that cause these symptoms often become **systemic** (move from the original point of infection to other living cells of the plant). Thus, virus diseases are different from foliar diseases caused by fungi and bacteria, which are usually relatively localized. Some viruses are located primarily in the phloem tissue; these are described in the section on vascular diseases.

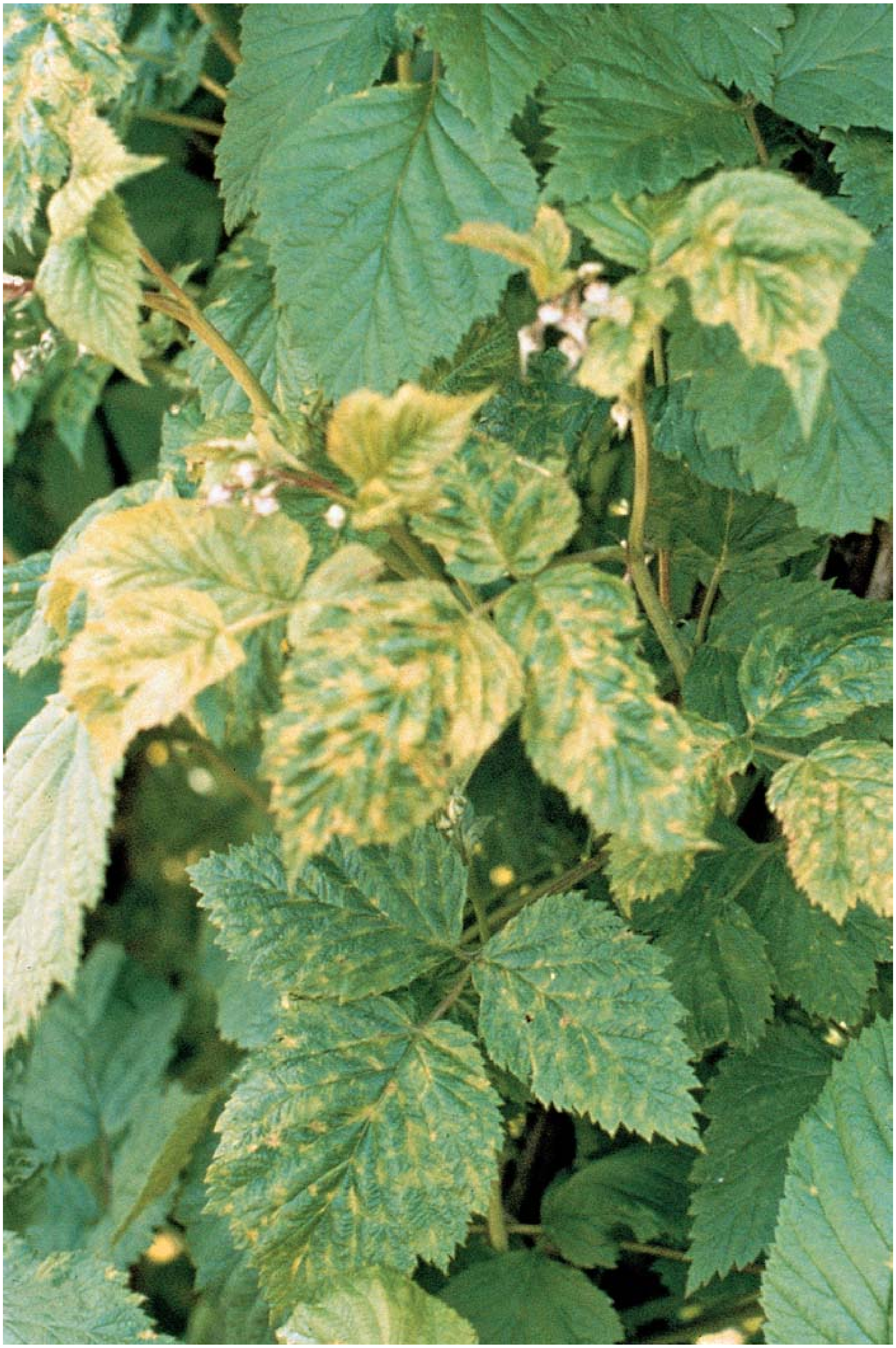


Figure 8.10. Red raspberry with foliar ring spots and line patterns caused by infection by *Tomato ringspot virus.*

Virus and Viroid Diseases

Plant Disease Profiles:

- **Barley yellow dwarf** (see Chapter 5, Disease Classic)
- **Cucumber mosaic**
- **Potato spindle tuber**
- **Soil-borne wheat mosaic**
- **Tobacco mosaic** (see Chapter 5, Disease Classic)
- **Tomato spotted wilt**

Virus diseases usually involve distortions and discolorations of leaves rather than the necrosis typical of many foliar problems. Some viruses are sap-transmissible, and many are spread by insects with piercing-sucking mouthparts. Because there are no chemical treatments for virus diseases, avoidance of the diseases and removal of infected plants are common disease management strategies (see Chapter 5).

Some viruses are sap-transmissible. Machinery, cutting tools, and people's hands can create wounds that allow viruses to infect cells in surface tissues. Wounds that allow virus entry also can be created by insects with piercing-sucking mouthparts (e.g., aphids, leafhoppers, whiteflies) or, less commonly, by insects with chewing mouthparts (e.g., beetles). There is no cure once a plant has become infected with a virus. Removal of plants suspected of being virus-infected will reduce the chances of disease spread.

Foliar Fungal Diseases with Diagnostic Signs

Some fungi and oomycetes produce distinctive signs that aid in diagnosis. In fact, the diseases they cause are named for these signs. Although predominantly found on leaves, they also may occur on herbaceous stems, flowers, fruits, and seeds. The examples described below include powdery mildew, downy mildew, rust, white rust, and smut.

POWDERY MILDEWS

Powdery mildews are different from most plant diseases because the pathogen remains mostly on the upper leaf surface. The white, septate hyphae produce numerous oval, white conidia (asexual spores), which give the surface of infected leaves a powdery look (Figure 8.11). These pathogens are **biotrophs** (**obligate parasites**) that absorb nutrients from epidermal cells via specialized hyphae called **haustoria**.

Figure 8.11. Powdery mildews are named for the powdery white growth of mycelium and conidia on plant surfaces.

Although apparently alike in appearance, the powdery mildew fungi are often quite host specific. The powdery mildew on a lilac bush is not caused by the same fungus as the powdery mildew of the nearby oak tree or the roses in the perennial garden. If they all have powdery mildew, it is probably because they are growing in the same favorable environment.

Another unusual feature of powdery mildew fungi is that the conidia can germinate in high relative humidity rather than requiring free water. Thus, powdery mildew can be quite severe even in years without a lot of rain as long as the air is humid. In general, powdery mildews are high-nitrogen diseases.

Injury from powdery mildew fungi usually takes some time to develop, and the economic impact depends on the host plant.

Powdery mildew of turfgrass in shady areas of a lawn is not usually of much concern. In contrast, even small amounts of powdery mildew on roses grown for cut flowers may be unacceptable, and the disease can be an important problem in greenhouse production.

Chasmothecia (**cleistothecia**), the overwintering fruiting bodies of powdery mildew fungi, are produced near the end of the growing season (Figure 8.12). When mature, they are very small, dark brown to black, round structures barely visible to the naked eye. Within them, asci form, releasing **ascospores** in the spring to initiate new infections. Thus, removal of debris infested with chasmothecia can reduce disease.

Figure 8.12. Life cycle of a powdery mildew fungus.

Powdery mildews are usually characterized by a superficial white mycelium, often on the upper surface of leaves. The hyphae absorb nutrients via haustoria. White conidia are the asexual, dispersal stage. Chasmothecia (cleistothecia), filled with asci and ascospores, are the sexual, survival stage. Humid conditions and application of excessive amounts of nitrogen favor the disease. Infested debris should be removed.

DOWNY MILDEWS

In contrast to powdery mildews, **downy mildews** are caused by oomycetes rather than ascomycetes. Like the powdery mildew fungi, these pathogens are biotrophs (obligate parasites) and tend to be highly host specific. Downy mildew pathogens are primarily dispersed through the air and infect aboveground parts of plants.

Downy mildews are caused by oomycete pathogens that are biotrophs (obligate parasites) and host specific. They are named for the downy growth of sporangia and sporangiophores, usually found on the lower surface of leaves. Oospores are the sexual and survival stage. Some downy mildews become systemic and cause distortions in plant growth. Leaf wetness and excessive nitrogen applications favor disease. Infested debris should be removed.

Downy mildews are named for the visible white to grayish brown downy growth (Figure 8.13) typically found on the lower side of leaves. With a microscope, it becomes apparent that the downy growth is composed of treelike **sporangiophores** with a **sporangium** at the tip of each branch (Figure 8.14). This structure is an adaptation of the pathogen for dispersal in air. The sporangiophores exit leaf tissue through stomata. In dicots, downy mildews are usually observed on the lower surface of the leaf, where most of the stomata are located (Figure 2.17).

Figure 8.13. Sporulation of a downy mildew pathogen, *Peronosclerospora sorghi*, on sorghum.

Figure 8.14. Sporangiophore and sporangia of a downy mildew pathogen.

Downy mildew begins as yellowish spots that can later become necrotic. **Grape downy mildew** can destroy both leaves and fruit. Downy mildew of tobacco, also called **blue mold**, causes significant losses in certain years as it is dispersed in air currents from south to north in the eastern half of the North American continent. Downy mildew is a common problem on cucurbits, brassicas (crucifers), hops, and snapdragon. Some downy mildew pathogens become systemic and disrupt the normal balance of plant growth regulators, causing **crazy top** of corn and **yellow tuft** of turfgrasses (Figure 8.15).

Figure 8.15. Yellow tuft, a systemic downy mildew disease of turfgrass, caused by *Sclerophthora macrospora*.

Leaf wetness favors infection by downy mildew fungi. The sporangia of many downy mildew pathogens produce swimming zoospores. In general, downy mildews are high-nitrogen diseases. Oospores are produced in infected plant tissue as sexual spores and survival structures, so removal of plant debris, when possible, helps reduce inoculum for the next year.

RUST

Rust diseases are named for the powdery orange spores in pustules that many rust fungi produce on infected plants (Figure 8.16). Rust diseases are commonly observed on asparagus, beans, brambles, cereal crops, coffee, corn, hollyhock, rose, snapdragon, turfgrasses, and many other hosts. Rust fungi are biotrophs (obligate parasites); most are quite host specific. They are unique in that many rust fungi require two unrelated host plants (**alternate hosts**) to complete the life cycle (thus, they are **heteroecious rusts**) and can produce up to five different spore stages. The life cycles of rust fungi can seem very complex, but the mycelium produces a logical progression of spore stages to accomplish genetic recombination and infection of both host species (see Chapter 2 for details).

Figure 8.16. Uredinia (A) and orange, powdery urediniospores (B) of *Puccinia graminis*, the fungus causing stem rust of wheat.

► **DID YOU KNOW?**

Wheat stem rust was a serious disease in ancient Greece and Rome. Rust was observed and recognized as early as the time of Aristotle (384–322 B.C.). The ancient Romans sacrificed red animals (dogs, foxes, and cows) to the rust god, Robigo (or Robigus), each spring during the festival called the Robigalia, in hopes that the wheat crop would be spared from the ravages of the rust. This festival was incorporated into the early Christian calendar as St. Mark's Day, or Rogation Day, observed on April 25.

> "No one can be a statesman who is entirely ignorant of the problems of wheat."
>
> —Socrates

Of special interest are the **urediniospores**. These are the powdery, rust- colored spores that give rust diseases their name. Urediniospores are called the **repeating stage** because they are the only rust spores that can cause new infections of the host species on which they were produced. Urediniospores can potentially cause devastating polycyclic epidemics.

Rust fungi that produce all five spore stages are **macrocyclic**. There are variations of this basic life cycle. Rust fungi that produce all spore stages except urediniospores (the repeating stage) are **demicyclic**. Rust fungi that produce only teliospores and basidiospores are **microcyclic**. In contrast to the heteroecious rust fungi that require two unrelated host plants, **autoecious** rust fungi produce all of their spores stages on a single host species.

When considering management of a rust disease, one must determine the host plants and, in the case of heteroecious rusts, which host, if any, has the urediniospores (repeating stage). For some heteroecious rust diseases, it is economical to remove one of the host plants for rust management. Does this eliminate the disease? The following examples show that the answer to this question depends on the rust fungus and on the economic value of the host plants.

In demicyclic rusts, such as cedar-apple rust, removal of either host, red-cedar (juniper) or apple/crabapple, theoretically eliminates the rust because there is no repeating stage. The spores from the junipers infect only apple, and the spores from the apple infect only junipers.

Common and Important Rust Diseases

Disease	Pathogen	Hosts (alternate hosts on separate lines)	Type
bean rust	*Uromyces phaseoli*	bean	autoecious, macrocyclic
cedar-apple rust	*Gymnosporangium juniperi-virginiae*	apple, crabapple red-cedar (juniper)	heteroecious, demicyclic
coffee rust	*Hemileia vastatrix*	coffee unknown alternate host	heteroecious

crown rust of oats and ryegrass	*Puccinia coronata*	oats buckthorn (*Rhamnus* spp.)	heteroecious, macrocyclic
hollyhock rust	*Puccinia malvacearum*	hollyhock	autoecious, microcyclic
soybean rust	*Phakopsora meibomiae,* *P. pachyrhizi*	soybean, other legumes unknown alternate host	heteroecious
stem rust of wheat and Kentucky bluegrass	*Puccinia graminis*	wheat, barley, and Kentucky bluegrass common barberry	heteroecious, macrocyclic
white pine blister rust	*Cronartium ribicola*	white pines currants, gooseberries (*Ribes* spp.)	heteroecious, macrocyclic

If the economically important host *does not* have the repeating stage, removal of the alternate host eliminates the disease because both the repeating stage and the teliospore survival stage are eliminated. For example, white pines can be protected from blister rust (*Cronartium ribicola*) by the removal of the alternate hosts, currant and gooseberry (*Ribes* spp.), on which the repeating stage is found. These hosts are the source of the basidiospores that initiate disease on pine. The rust does not spread from pine to pine.

If the host of economic importance *does* have the repeating stage, disease management is more challenging. Urediniospores are usually killed by winter conditions, but they may potentially still cause epidemics. For example, urediniospores can survive in the protected environment of a greenhouse until a new crop is established. Carnation, chrysanthemum, and geranium are all repeating-stage hosts of their specific rust fungi, so rust epidemics can occur in these crops even in the absence of the alternate hosts.

In the early twentieth century, an extensive barberry eradication program was implemented to remove the alternate host of wheat stem rust (*Puccinia graminis*) and protect U.S. wheat crops. Unfortunately, urediniospores can overwinter on wheat in the southern United States and northern Mexico and then move northward each year via the **Puccinia pathway** (named for the common rust genus *Puccinia*) as far as the wheat fields of Canada (Figure 8.17). Although these rusts have not been eliminated, the absence of the alternate host still breaks the sexual cycle and reduces genetic recombination in the pathogen. It also delays epidemics and reduces the primary inoculum from barberry bushes.

► **DID YOU KNOW?**

Barberry was the primary source of new stem rust races in the United States before an eradication program was begun in 1918, lasting through 1975. Over 100 million barberry bushes were eradicated.

Figure 8.17. The Puccinia pathway. Each year urediniospores of rust fungi are carried by winds from Mexico and the southern United States across North America to Canada.

DISEASE CLASSIC

Stem Rust of Wheat and Barley

Hosts barberry (aecial host); wheat and barley (telial hosts)

Pathogen *Puccinia graminis*

Symptoms and Signs Pustules containing urediniospores (orange) and teliospores (black) on cereal hosts; pycnia and blisterlike **aecia** on barberry

Disease Cycle *P. graminis* is a macrocyclic, heteroecious rust. Basidiospores of the fungus infect only barberry leaves, where they produce pycnia in which a **dikaryotic** mycelium is formed. Dikaryotic aeciospores are produced in aecia on the lower surface of infected barberry leaves. The aeciospores infect the cereal hosts. Seven to 14 days after infection, the first pustules of powdery, orange urediniospores are produced. The urediniospore is the repeating stage—a urediniospore can reinfect the cereal host, so that more urediniospores are formed in the infected tissue, causing polycyclic epidemics in wheat and barley fields. Later in the season, the mycelium in infected cereal hosts produces **telia**, in which black teliospores are formed. The teliospore is the survival stage of the fungus. The dikaryotic nuclei of teliospores fuse and undergo meiosis to produce haploid basidiospores, which infect only barberry.

Management A massive barberry eradication campaign in North America eliminated the completion of the sexual cycle and greatly reduced the number of common races of the pathogen. Unfortunately, urediniospores continue to move in wind currents from the south to the north along the Puccinia pathway, but the amount of inoculum is reduced and its arrival is delayed in northern areas. Genetic resistance is crucial in disease management.

Significance It is estimated that more than $5 billion is lost due to cereal rusts (leaf rust, stem rust, and stripe rust) each year. Ug99, a new race of the stem rust pathogen, represents a threat to global wheat production. For more information, see Chapter 9.

Plant Disease Profiles: Stem rust

Removal of the alternate host is not an option for autoe-cious (single-host) rusts. Both autoecious and heteroecious rust diseases may be managed using the same methods used for other foliar fungal diseases: genetic resistance, minimizing leaf wetness, removal of infested debris, proper fertility levels, and fungicide applications. In general, rust diseases are high-nitrogen diseases.

Management of Rust Diseases

demicyclic rust

- No urediniospores (repeating stage) are produced.
- Removal of **either** host eliminates disease.

macrocyclic rust

- If urediniospores are produced on the **less** valuable host, removal of this host eliminates the disease.
- If urediniospores are on the **more** valuable host, removal of the alternate host eliminates disease
    - **unless** urediniospores can survive and infect a crop (e.g., via Puccinia pathway, greenhouses, or other mild environments).

autoecious rust

(also heteroecious rust in which urediniospores are present on a valuable host and can survive)

- Standard foliar disease management is the only option.

Rust fungi are basidiomycetes that may require two unrelated host plants to complete their life cycles and may produce up to five spore stages. They are biotrophs (obligate parasites) and host specific. Rust diseases are named for the powdery orange urediniospores (repeating stage) that can result in polycyclic epidemics. Heteroecious rust fungi require two different host plants. Removal of the alternate host is sometimes used to manage heteroecious rusts. Autoecious rust fungi complete their life cycles on a single host species and are managed as typical foliar fungal pathogens.

White rusts are named because of the powdery white sporangia that are produced in leaf pustules similar to those of the orange rusts (Figure 8.18). Unlike the orange rusts, which are caused by basidiomycetes, the white rusts are caused by oomycetes in the genus *Albugo*. The sporangia produce zoospores when exposed to water, and the sexual, survival spores are oospores. White rust occurs on *Amaranthus* (pigweed), brassicas/crucifers, spinach, sweet potato, and some ornamentals. *Albugo* species are biotrophs (obligate parasites) and host specific. Like the downy mildews, white rust is air-dispersed and affects aboveground plant parts. Affected plants may be severely distorted and have reduced seed production in certain seed crops. White rust may be seedborne. Management of white rust includes planting of clean seed and disposal of infested plant debris.

Figure 8.18. White rust, caused by *Albugo* sp., on a morning-glory leaf (*Ipomoea* sp.).

White rust is caused by oomycetes in the genus *Albugo*. The disease is named for the pustules of white powdery sporangia that form in infected leaves. Zoospores are produced for infection. Like downy mildews, *Albugo* spp. are biotrophic, host specific, and adapted for air dispersal. The survival spores are oospores that may form in infected tissue. Use of clean seed and disposal of infested debris reduce disease.

SMUT

Smut diseases are named for the black powdery **teliospores** that are typically produced by the smut fungi. Each cell of the mycelium forms a thick, dark wall and becomes a teliospore that is both the sexual and survival stage of the smut fungus (see Chapter 2). These masses of "dirty" spores can be found in bulbs (onion smut), flowers (anther smut), leaves (stripe smut of turfgrasses), seeds (loose smut and stinking smut of wheat), stems, and roots.

Symptoms of smut infection vary. In some smut-infected cereals, symptoms may not be apparent until the harvest of the teliospore-filled seeds (Figure 8.19). Corn smut produces large, edible **galls** filled with teliospores, which are valued by gourmets in the United States and Mexico. Corn smut is a common disease in sweet corn plantings. (More information about corn smut is in the section of this chapter on gall diseases.)

Figure 8.19. Cloud of smut teliospores during wheat harvest.

Plants with leaf or flower smut infections may be stunted, yellowed, or malformed. The presence of numerous teliospores in infected tissues can confirm the diagnosis of smut. White smuts of the genus *Entyloma* are common on ornamental plants such as calendula, composites (such as aster and daisy), and dahlia. The teliospores are clustered in light-colored blisters. The white color is from the **sporidia** produced from teliospores. Management of smut diseases typically involves seed treatments for cereal smuts and removal of infected plants and infested debris for ornamentals and vegetables.

► DID YOU KNOW?

When contaminated by teliospores of the stinking smut (common bunt) pathogens, wheat flour acquires an off-color and the smell of rotting fish. Although not toxic, it is unappealing. It is said that gingerbread was invented to mask the color and flavor of contaminated flour. Mathieu Tillet (1714–1791), a French farmer, made some important early observations about the nature of the disease. He demonstrated that wheat contaminated with smut spores later grew into plants with masses of smut spores instead of healthy grain. Tillet’s name is now inexorably linked to stinking smut, which is caused by *Tilletia* species.

DISEASE CLASSIC

Stinking Smut (Covered Bunt) of Wheat

Host wheat

Pathogens *Tilletia tritici* and *T. laevis* (basidiomycetes)

Symptoms and Signs Symptoms are not obvious, but seeds are filled with smelly black teliospores at harvesttime.

Disease Cycle Teliospores produce haploid basidiospores called sporidia. The mycelia from germinating sporidia form distinctive H-shaped structures to produce the dikaryotic mycelium necessary for infection of germinating wheat plants. The mycelium grows internally along with the plant, eventually replacing seed cells with teliospores. At harvest, the teliospores contaminate healthy seed and nearby soil.

Management Stinking smut is a mono-cyclic disease and is primarily managed with seed treatments.

Significance From the late 1800s until the 1930s, stinking smut was a devastating disease of wheat, particularly in winter wheat grown in the Pacific Northwest of the United States. In 1890, between 25 and 50% of the Kansas wheat crop was lost to stinking smut. Farmers were sometimes killed when sparks from metal harvesting equipment triggered explosions of clouds of smut spores.

Plant Diseases Profiles: Stinking smut

Smut diseases are caused by basidiomycetes that typically produce masses of black powdery teliospores. Teliospores form from the individual cells of the mycelium. They are the sexual and survival spores. Management varies depending on the type of smut disease.

What are vascular diseases?

Some pathogens invade only the xylem or phloem of the plant. Xylem pathogens cause vascular wilts and interfere with water and mineral transport. Typical symptoms are wilt, scorch of leaves, stunting, and yellowing. Phloem pathogens interfere with the transport of photosynthates in the plant. Typical symptoms are overall yellowing and stunting but usually not wilting.

Vascular Wilts

Vascular wilts are xylem diseases. They are characterized by wilting, but this nonspecific symptom also may be due to drought or some problem in the roots. A discoloration of the active xylem is an important diagnostic symptom associated with vascular wilt infections. The discoloration can be observed by slicing away the outer stem or twig tissues (Figure 8.20); a cross-sectional cut should reveal a ring of brownish outer xylem. If the host is a tree or mature plant, only part of the xylem may become infected, leading to wilt on only one side (Figure 8.21). In woody plants, a vascular wilt may show up as **flagging**, which describes the wilting of leaves on individual branches. Most vascular wilts begin as root infections, so leaves may become yellowed and wilted from the bottom of the plant upward over time. If bacteria are the cause of the vascular wilt, the cut stem of an infected plant may yield strands of sticky bacteria (ooze) (Figure 1.6B).

Figure 8.20. Streaking of vascular tissue due to infection by *Verticillium* sp.

Figure 8.21. Sugar maple with branch dieback due to Verticillium wilt. Note the one-sided symptoms.

Symptoms and Signs of Vascular Diseases	Disease Type
vascular wilt pathogens (ascomycetes and	xylem diseases

bacteria) • wilt, flagging, stunting, yellowing • discoloration of active xylem • one-sided wilt, in some cases • wilt progressing in plant from bottom up, if pathogen is soilborne • ooze in xylem if a bacterial pathogen	
xylem-limited fastidious bacteria • wilt, scorch, stunting, yellowing	xylem diseases
phloem-limited fastidious bacteria (including phytoplasmas and spiroplasmas) • stunting, yellowing, witches' brooms	phloem diseases
viruses • stunting, yellowing	usually phloem diseases

Insects are vectors of some bacterial vascular wilt pathogens. As insects feed, they make wounds. These can be contaminated by their feces, which carry bacteria, so that the pathogens gain entry to the xylem. *Erwinia tracheiphila* causes **cucurbit bacterial wilt** and is transmitted by cucumber beetles. *Erwinia stewartii* (syn. *Pantoea stewartii*) causes **Stewart's wilt** of corn and is spread by corn flea beetles. Management of the vectors of these pathogens helps prevent disease.

Ralstonia solanacearum (previously called *Pseudomonas solanacearum*) causes **southern wilt** of a number of economically important plants in the United States, including tomato, potato, and tobacco. Worldwide, one race of this pathogen causes bacterial wilt in more than 300 different plant species, and another race causes **Moko disease**, a bacterial vascular wilt of banana.

Two important vascular wilts are caused by soilborne fungi. **Verticillium wilt** is caused by several *Verticillium* species that produce a white mycelium with either dark **microsclerotia** or melanized hyphae as survival structures, depending on the species. The genus *Verticillium* is named for the whorled pattern of short, spore-producing structures around each conidiophore, a pattern described as verticillate. *Verticillium* spp. have wide host ranges and can survive in the soil for many years.

► **DID YOU KNOW?**

The peppermint industry was centered in the midwestern United States in the mid- to late 1800s, when Verticillium wilt emerged as a destructive disease there. Peppermint acreage has increased in the western states since the 1930s, as Verticillium wilt intensified in the Midwest. Since the 1960s, half of the U.S. peppermint acreage has been in Oregon. The spread of *Verticillium* spp. through regions of Idaho, Oregon, and Washington is now forcing peppermint growing to move into uninfested areas of these states and to other western locations. Peppermint is grown for the oil it yields. Oil yields and stand longevity are reduced as wilt intensifies in a field.

Fusarium wilt is caused by a pink to yellow fungus, *Fusarium oxysporum*. It produces three kinds of spores. The **macroconidia** are spindle-shaped or fusoid, giving the fungus its name. (See the section on Fusarium diseases later in this chapter.) Macroconidia are the primary infectious spores. The **microconidia** are smaller and have only one or two cells. They are important in the movement of the fungus within the xylem vessels. The **chlamydospores** are thick-walled survival spores that have various shapes and can be formed from hyphal cells or individual cells of the macroconidia (Figure 2.20).

The species *F. oxysporum* has been divided into a number of special forms, or ***formae speciales*** (f. sp.), based on their host range. For example, *F. oxysporum* f. sp. *lycopersici* causes Fusarium wilt of tomato, whereas *F. oxysporum* f. sp. *cubense* causes Fusarium wilt (**Panama disease**) of banana. Because the symptoms of Verticillium wilt and Fusarium wilt are essentially identical, accurate diagnosis is important to determine which plants can be safely planted in soil infested by these pathogens. Fusarium wilt threatens plants only if the appropriate *forma specialis* is present, but Verticillium wilt can affect hundreds of different plant species.

	Verticillium wilt	**Fusarium wilt**
fungal colony color	white	often colored, orange to bluish to purple
asexual spores	tiny conidia on verticillate structures	curved macroconidia, smaller microconidia
survival structures	microsclerotia or melanized hyphae	chlamydospores
host range	wide host range	host range varies: most *formae speciales* infect plants in only one

genus or a few genera

Dutch elm disease and **oak wilt** are two vascular wilt diseases caused by fungi that are vectored by insects. The Dutch elm disease pathogen is spread by adult elm bark beetles that emerge from larval galleries under the bark of dying elm trees (Figure 8.22). The pathogens (*Ophiostoma novo-ulmi* and *O. ulmi*) produce sticky conidia on **synnemata** (asexual fruiting bodies) (Figure 8.23) in the beetle galleries. Beetles carry some spores on them when they emerge and fly to a healthy elm to feed on the young twigs in the crown of the tree. Thus, the pathogen never has a soilborne stage. The beetles efficiently transfer the pathogens from a dying elm to a new elm host.

Figure 8.22. Egg gallery of the European elm bark beetle. Eggs are laid in the maternal tunnel, under the bark and parallel to the grain of the wood. Synnemata of *Ophiostoma ulmi* are produced in the larval tunnels, which are perpendicular to the maternal tunnel and produced by the feeding larvae.

Figure 8.23. Light microscope (left) and scanning electron microscope (right) views of synnemata of *Ophiostoma ulmi*, one of the fungi causing Dutch elm disease. The conidia stick to adult elm bark beetles as they emerge from egg galleries (see Figure 8.22).

Oak wilt is caused by a related fungus, *Ceratocystis fagacearum*, which produces fungus pads under the bark of infected oak trees. The odor from the fungus pads attracts various insects that feed on the fungus pad and pick up spores. The vector relationship is not as specific and efficient as in Dutch elm disease because the insects may or may not travel directly from the fungus pad to a healthy oak.

When plants of the same species grow near each other, their roots may graft below ground. Vascular wilt pathogens may move through these root grafts and infect new hosts, a transmission mechanism for Dutch elm disease that resulted in the loss of millions of elm trees planted for shade along city streets throughout Europe and North America over the past century.

Both Dutch elm disease and oak wilt cause flagging and eventually death of the tree. Symptoms vary depending on the means of infection. If infection is through root grafts with a diseased neighboring tree, the symptoms of flagging and branch death are often quite dramatic and severe. Symptoms resulting from insect transmission begin in the crown of the tree and may involve the flagging of one or a few small branches. These branches can sometimes be pruned to save the tree. Severely infected elm and oak trees should be removed to reduce spread by vectors and transmission through root grafts. Elms with genetic resistance to Dutch elm disease are now available.

DISEASE CLASSIC

Dutch Elm Disease

Hosts American and European elms

Pathogen *Ophiostoma novo-ulmi* and *O. ulmi* (ascomycetes)

Symptoms and Signs Wilting, flagging of leaves on branches, brown discoloration of active xylem

Disease Cycle Elm bark beetles vector the fungi. The beetles form galleries under the bark of dying elms. In these galleries, the pathogen produces sticky conidia on synnemata. The conidia stick to emerging beetles, and, as they feed on small twigs in the crown of the tree, spores are introduced to the xylem. An infected tree can be a source of inoculum that can infect a neighboring tree through root grafts.

Management Diseased and dead trees should be removed to reduce the reproduction of elm bark beetles. Early infections in the crown of the tree can be removed by pruning. Fungicide injections are useful for managing the disease in valuable trees. Elm breeding programs for genetic resistance have been successful.

Significance The pathogens and the European elm bark beetle were imported to North America from Europe on several occasions in elm logs. Dutch elm disease is a classic example of the dangers of exotic pathogens and the dangers of monoculture. Millions of urban trees have died as infections spread through root grafts along elm-lined streets.

Plant Disease Profiles: Dutch elm disease

► DID YOU KNOW?

Over 40 million American elm trees have been killed by Dutch elm disease, and today it is still the most destructive disease of shade trees in the United States. Note that the disease has killed American elms in the United States—not "Dutch elms." The disease is named to honor the Dutch women scientists who studied it in the first half of the 20th century. A large population of European elms exists in Australia. An elm bark beetle vector is present in Australia, but so far the *Ophiostoma* pathogens have not been introduced there.

Xylem-Limited Fastidious Bacteria

Organisms such as *Xylella fastidiosa* invade the xylem and cause water-stress symptoms similar to those of the more common vascular wilts (see Chapter 3). These bacteria can be cultured only on complex media, so they are called **fastidious**. Symptoms include stunting, yellowing, wilting, and plant death. Scorch is another common symptom in which the edges of the leaves are necrotic (Figure 8.24). The marginal necrosis on leaves is bordered by a halo of yellow to reddish cells.

Figure 8.24. Elm leaf scorch, caused by *Xylella fastidiosa*. Note the undulating marginal necrosis with a chlorotic halo.

These pathogens are not sap-transmissible, so they are not spread by touching or by machinery and tools. They are vectored by xylemi feeding insects such as sharpshooters (Figure 3.14). *X. fastidiosa* causes Pierce’s disease of grapevine and bacterial scorch of shade trees. Management of these diseases is difficult. In grape plantings, removal of infected plants is often recommended to reduce the spread of Pierce’s disease.

Phloem-Limited Fastidious Bacteria and Viruses

Plants infected by **phytoplasmas**, **spiroplasmas**, and other phloem-inhabiting bacteria generally exhibit overall stunting and yellowing, but not usually wilting (see Chapter 3). Some of the pathogens have been isolated on complex media (thus they are fastidious bacteria), but most of the phytoplasmas have yet to be cultured. Phytoplasmas and spiroplasmas can disrupt the normal balance of growth regulators of plants and cause various distortions, including the proliferation of stems from a single point known as a witches’ broom (Figure 8.25). These pathogens are not sap-transmissible. Phytoplasmas and spiroplasmas are vectored by leafhoppers. Other phloem-inhabiting bacteria are vectored by psyllids and some other insects. Lethal yellowing of coconut palm and aster yellows, a disease of many flower and vegetable crops, are important phytoplasma diseases.

Figure 8.25. Ash yellows on a green ash tree, caused by a phytoplasma. Note the witches' broom on the trunk.

The fastidious bacteria are sensitive to certain antibiotics such as tetracyclines. Remission of symptoms after application of appropriate antibiotics may be used for preliminary diagnosis and for management in high value plants. In most cases, removal of infected plants is recommended to reduce disease spread.

Many of the diseases caused by phloem-limited bacteria were originally considered to be caused by "yellows" viruses because no pathogen could be isolated and symptoms were similar to those of virus diseases. Yellows diseases are caused by viruses that invade the phloem of host plants and disrupt the flow of photosynthates. These viruses often do not invade surface tissues and therefore are not sap-transmissible. They are commonly spread by insect vectors (e.g., aphids) that feed in the vascular tissues of the plants. Insecticides are sometimes used for vector control, but management of these diseases by resistance or removal of infected plants is often more economical and effective.

Vascular Diseases

Introduction to Fastidious Bacteria
Plant Disease Profiles:
  fungal vascular wilts
    Dutch elm disease (see Chapter 8, Disease Classic)
    Fusarium wilt of watermelon and other cucurbits

- **Verticillium wilt** (see Chapter 10, Disease Classic)

- **bacterial vascular wilts**
  - **Bacterial leaf scorch**
  - **Stewart's wilt** (see Chapter 9, Disease Classic)
- **phloem diseases**
  - **Barley yellow dwarf** (see Chapter 5, Disease Classic)
  - **Lethal yellowing of palms**

Vascular pathogens invade the phloem or the xylem. Vascular wilts are xylem diseases and may be caused by bacteria or fungi. Insects are often vectors of the common bacterial vascular wilts. Bacterial ooze is usually abundant in infected stems. A ring of vascular discoloration is a common symptom of vascular wilts. Soilborne fungi cause Fusarium wilt and Verticillium wilt. Dutch elm disease and oak wilt are fungal vascular wilts with insect vectors and no soilborne stage. *Xylella fastidiosa* is a bacterium that resides in the xylem, causes wilt diseases, and is vectored by xylem-feeding insects such as sharpshooters. Fastidious bacteria, phytoplasmas, spiroplasmas, and viruses can cause phloem diseases. Common symptoms are stunting and yellowing. Management of vascular diseases is difficult and usually involves the use of resistant cultivars, when available, and removal of infected plants to reduce disease spread.

What are cankers?

Cankers are symptoms of some branch, stem, and twig diseases. Cankers are usually dry, necrotic, localized lesions on the stem, branch, or trunk. The lesions are often sunken, but a rim of callus may form in response to the infection. It is common to observe fruiting bodies in cankers caused by fungal pathogens. The tissues involved are the tissues to the outside of the xylem, in particular the phloem and vascular cambium in woody plants. Thus, cankers are not wood decay diseases. If the pathogen invades the tissues below the bark and encircles a stem or branch, all plant tissue above the canker will die. For example, when a canker pathogen **girdles** the trunk of a tree, the tree will die.

Cankers

- diseases of branches, stems, and twigs
- dry, necrotic, localized lesions, often sunken, but may be surrounded by raised callus
- phloem and vascular cambium below bark are killed
- caused by fungi or bacteria
- pathogen entry is through wounds

Cankers

Plant Disease Profiles:

bacterial

Fire blight (see Chapter 3, Disease Classic)

Citrus canker (see Chapter 10, Disease Classic)

fungal

Leucostoma canker of stone fruits

As described previously, cankers sometimes develop in plants with **anthracnose** diseases. After invasion of the more vulnerable tissues of leaves, flowers, and fruits, the pathogen may continue to spread and eventually cause cankers in stems or twigs. Examples include **black rot** of apple, **black spot** of rose, and anthracnose diseases of dogwood, maple, oak, and sycamore. Foliar fungicide applications can protect the leaves from the initial infections that lead to cankers. They are usually not necessary except in very wet years or in the case of an exotic pathogen. For example, dogwood anthracnose is a destructive disease because the native dogwoods have little resistance to *Discula destructiva*, a fungus that was accidentally introduced to North America.

Both bacteria and fungi cause canker diseases (Figure 8.26). Ascomycetes cause most of the fungal cankers. Cankers under the bark of woody plants provide protected survival sites for these pathogens during the winter months.

Figure 8.26. Leucostoma canker is a fungal disease and is one of the most important diseases of stone fruits in the world. Note the amber gum oozing from the infected tissues. A pruning cut (arrow) in the center of the canker served as the infection court.

One of the most destructive canker diseases is **chestnut blight,** caused by the fungus *Cryphonectria parasitica* (Figure 8.27). Because the pathogen was accidentally introduced from Asia to a chestnut population with little or no resistance, the American chestnut is no longer a dominant species throughout its natural range in the Appalachian Mountains. Other common canker diseases are **Dothichiza canker** of Lombardy poplar, **Hypoxylon canker** of aspens, **Nectria canker** of hardwoods, and **Scleroderris canker** of red and jack pines. It may be possible to diagnose a canker disease by examining the fruiting bodies of the pathogen, which may form near the leading edge of a canker. For example, salmon-colored **sporodochia** (asexual fruiting bodies) and red **perithecia** (sexual fruiting bodies) may be found in Nectria cankers (Figure 8.28). Orange fruiting bodies are common in chestnut blight cankers.

Figure 8.27. Chestnut blight canker.

DISEASE CLASSIC

Chestnut Blight

Hosts American and European chestnuts

Pathogen *Cryphonectria parasitica* (an ascomycete)

Symptoms and Signs The cankers are not very obvious on most trees. Brownish orange fruiting bodies (perithecia and pycnidia) of the fungus push through the bark.

Disease Cycle Ascospores and conidia are dispersed by air and water to new infection sites. Spores also may travel on insects. Infection usually requires wounds from storms or insect feeding.

Management Diseased and dead trees should be removed to reduce inoculum production, but removal is not practical in forests. Pruning of cankers is helpful in chestnut orchards. Hypovirulent strains of the chestnut blight fungus are used as a biological control (see Chapter 11). Scientists are selecting trees for genetic resistance through breeding programs.

Significance The chestnut was once a dominant tree in the Appalachian Mountains, but millions of trees were killed following accidental introduction of the pathogen, which probably originated in Asia. Chestnut was an important source of timber, nuts, and tannins for the leather industry.

Figure 8.28. Nectria canker (coral spot) on a hardwood tree. Note the fruiting bodies emerging through the bark.

The severity of a canker disease depends on the size, number, and location of wounds. Usually the bark protects the soft phloem and vascular cambium tissues, but this protective barrier can be breached by wounds caused by storms, insects, or human activities such as pruning. Stress factors such as compacted soil, drought, and nutrient deficiencies reduce defense reactions by the host. Severity also depends on the genetic interaction between the host and the pathogen. Exotic pathogens are more likely to cause tree death than native pathogens, to which the host probably has some resistance.

Native pathogens can be a threat to plants that are moved out of their natural environment. For example, the Colorado blue spruce, adapted to a dry habitat, is more susceptible to canker diseases when planted in more humid regions. Native pathogens also may cause cankers on woody ornamentals that originated in Asia or Europe. Monocultures of either native or exotic species favor canker pathogens because inoculum is more likely to successfully land on another susceptible host.

When practical, pruning of cankers removes sources of inoculum and minimizes disease spread in the coming season. Woody landscape plants and fruit trees are routinely pruned to reduce bacterial and fungal canker diseases. Pruning tools should be disinfested to prevent introduction of pathogens from contaminated tools into pruning wounds. Pruning cuts must be well beyond the border of the apparent canker to remove tissue colonized by fungal and bacterial pathogens that may not be apparent on the surface.

Cankers are symptoms of branch, stem, and twig diseases of woody plants in which the phloem and vascular cambium are killed below the bark. Once a canker has girdled a branch or trunk, the branch or trunk will die. Cankers may be caused by bacteria or fungi. The severity of a canker disease depends on the environment, the health of the tree, wounds, and the

genetic interaction between host and pathogen. Removal of the cankers and prevention of wounds are the most important management practices.

What are galls?

Galls or tumors are caused by a variety of pathogens. These pathogens either produce plant growth regulators themselves or induce changes in the amount or balance of growth regulators that the host plants normally produce. Galls are usually a result of an increase in both the size of cells (**hypertrophy**) and the number of cells (**hyperplasia**).

Galls

- abnormal, somewhat spherical growths on leaves, stems, or roots
- caused by bacteria, fungi, insects, nematodes, or viruses

Black Knot

Apiosporina morbosa is an ascomycete that infects *Prunus* species (cherry and plum). It causes easily visible crusty black galls and cankers on twigs and branches (Figure 2.23). The pathogen can kill a tree if it completely girdles the trunk. Pruning is the most common management practice.

Cedar-Apple Rust

Gymnosporangium juniperi-virginianae is a heteroecious, demicyclic rust fungus. When aeciospores from apple or crabapple trees infect junipers (red-cedar), galls are produced (Figure 8.29A). In warm, rainy spring weather, orange tendrils of teliospores emerge from the gall and produce **basidiospores**. Because basidiospores from the junipers are the only source of infection of the apple or crabapple trees (Figure 8.29B), removal of juniper trees near apple orchards can reduce this monocyclic disease. All junipers within an 8-kilometer (5-mile) radius must be removed to eliminate the disease. Apple, crabapple, and junipers with genetic resistance to this rust are available.

DISEASE CLASSIC

Cedar-Apple Rust

Hosts apple and crabapple (aecial hosts); red-cedar (juniper) (telial host)

Pathogen *Gymnosporangium juniperi-virginianae*

Symptoms and Signs Small brownish galls form on juniper branches. In spring, they produce orange, gelatinous tendrils. Pycnia form on the upper surface of apple leaves, and reddish orange, blisterlike aecia form on their lower surface. Fruits may also be infected.

Disease Cycle Basidiospores are formed on germinating teliospores of the expanded juniper galls. The basidiospores infect apple leaves to produce pycnia. Aeciospores are eventually produced in blisterlike aecia on the lower surface of the apple leaf. The aeciospores infect only juniper. The pathogen is a heteroecious, demicyclic rust (it does not form urediniospores). The disease is monocyclic.

Management Because the disease is monocyclic, fungicide applications in orchards can be timed to protect trees during the short period when basidiospores are released. Many crabapples have genetic resistance to the disease. Removal of nearby junipers reduces the disease in apple orchards.

Significance Junipers near apple orchards are routinely removed. Landscapers should consider the risk of disease before planting crabapples and junipers near each other. The disease is not very damaging, except in wet years. Infection of apple fruit is the primary economic impact.

Figure 8.29 A, Telial gall of cedar-apple rust on red-cedar (juniper). The orange spore horns have withered after basidiospore production. B, Cedarapple rust symptoms on apple. The infections were caused by basidiospores produced on the telial gall, resulting in pycnia. Later, aeciospores are produced from aecia on the lower leaf surfaces.

Clubroot of Brassicas (Crucifers)

Plasmodiophora brassicae causes root galls (Figure 2.18). It is an economically important, soilborne, fungus-like pathogen of cabbage, broccoli, cauliflower, and related plants. The resting spores are released from infected roots as they decay, so the early removal of the roots of infected plants helps reduce primary inoculum for the next season. Manure from animals that have eaten infected roots can be a source of inoculum if spread on agricultural land. Addition of lime to raise the soil pH to 7.2 or above reduces spore germination.

Corn Smut

The large galls of *Ustilago maydis* can form in any growing point of a corn plant (Figure 2.12). Sweet corn is more susceptible than field corn. Mature galls are filled with the teliospores of the smut fungus. The galls should be removed before the spores are released to reduce inoculum of this monocyclic disease for the next season.

DISEASE CLASSIC

Common Smut of Corn

Host corn (maize)

Pathogen *Ustilago maydis* (a basidiomycete)

Symptoms and Signs Large galls filled with mycelium form on corn plants. Over time, each cell of the mycelium develops into a black teliospore, and mature galls become filled with a powdery mass of these spores.

Disease Cycle Within the teliospore, the nuclei fuse and undergo meiosis. Teliospores form haploid basidiospores (sometimes called sporidia), which serve as the primary inoculum. Only a dikaryotic mycelium, formed by the fusion of the hyphae from two germinating basidiospores, can infect the corn plant. Any young growing point of the plant can be infected. The fungus stimulates the plant to produce large galls of corn tissue filled with the dikaryotic mycelium. The fragile tissue of a mature gall can be easily broken open to release the teliospores for infections in the next season. Corn smut is a monocyclic disease.

Management Hand picking of galls before they break open will reduce the level of inoculum in the following season. Genetic resistance is the primary means of management in commercial production.

Significance Sweet corn is more susceptible to corn smut than field corn. The disease is primarily a problem when galls form on the ear. Smut galls are edible (Fig. 8.30) and are considered a delicacy by many gourmets.

Plant Disease Profiles: Common smut of corn

Figure 8.30. Portion of a mural painted by Guillermo Lourdes in the city of Durango, Mexico, showing the importance of corn in Meso-american culture. Note corn smut on the ears of corn. From a painted copy by Rudy Cruz.

Crown Gall

Agrobacterium tumefaciens is one of the most well-studied of bacterial plant pathogens. It is a natural genetic engineer that transfers genes for the production of auxin and cytokinin to the plant genome via the Ti (tumor-inducing) **plasmid**. Crown gall is a common disease of many woody ornamentals, including *Euonymus* and rose (Figure 3.11). This bacterium has a wide host range and can inhabit the rhizospheres of a wide variety of herbaceous and woody plants, including weeds, for many years. Crown gall bacteria often infect plants through small wounds near the soil line and in areas of freeze damage. Planting equipment, pruning tools, mowers, and string weeders are common causes of wounds on woody plants that may be invaded by crown gall bacteria. Care must be taken to disinfest pruning tools to avoid spreading the bacteria.

Exobasidium Galls

Exobasidium vaccinii causes galls in the spring on aboveground parts of ericaceous plants, including azalea, blueberry, cranberry, and rhododendron. Basidiospores are produced on the surface of the gall, giving them a powdery look. The disease is not usually economically important. On landscape plants, galls should be picked and destroyed before they turn powdery.

Root Knot

Secretions from the feeding female root- knot nematode (*Meloidogyne* spp.) contain auxinlike molecules that induce small root galls (Figure 4.2). The galls can be numerous when nematode populations are high.

Galls

Plant Disease Profiles:

- **Black knot**
- **Common smut of corn**
- **Crown gall** (see Chapter 3, Disease Classic)
- **Root-knot** (see Chapter 4, Disease Classic)

See also

- **Clubroot** (see Chapter 2, Disease Classic)

Galls form when a pathogen produces plant growth regulators directly or disrupts their natural balance in the plant. Bacteria, fungi, nematodes, viruses, and many kinds of insects and related organisms can cause plant galls to form. Management of gall diseases varies considerably, depending on the pathogen and host plant.

What are root rots?

The roots of plants can be damaged by environmental extremes such as drought, freezing, or poor drainage. Once the tissues have been killed, saprophytes colonize them as part of the decay process. There also are situations in which pathogens invade healthy plant roots and cause diseases known as root rots. These are particularly frustrating diseases because much of the damage occurs before symptoms are apparent above ground. In addition, it is generally difficult to effectively and economically deliver fungicides to the roots of plants even if the disease is identified in its early stages. Root rots are more likely to develop when roots are wounded or following infection caused by migratory, endoparasitic nematodes such as lesion nematodes (*Pratylenchus* spp.). Many root pathogens survive well in the absence of their host plants and are categorized as soil inhabitants with wide host ranges. This means that crop rotation to nonhost plants may not be effective in reducing root pathogen populations, so more costly methods of heat or chemical soil treatments may be used before a new planting.

DISEASE CLASSIC

Take-All Root Rot

Hosts small grains, bentgrasses

Pathogen *Gaeumannomyces graminis* (an ascomycete)

Symptoms and Signs In cereals, prematurely ripe seed heads appear in circular patches. In close-mown creeping bentgrass (on golf putting greens, for example) the turf declines and dies in circular patches. In both situations, roots will appear dark brown to black and rotted. Dark, ectotrophic hyphae and mycelial mats form on roots and stolons. Dark perithecia also may be observed.

Disease Cycle Ascospores are produced in perithecia, but they are not important in the development of the disease in the field. The ectotrophic hyphae grow from infested debris or infected plants to the roots of new plants.

Management Root surface pH can be reduced with ammonium sulfate or other chemicals. Crop rotation is effective for management of take-all in annual crops. When crops are not rotated, a natural biological control known as take-all decline can develop over time (see Chapter 11). Fungicides are used to manage the disease in turfgrasses.

Significance Take-all is a worldwide problem. The pathogen is a parasite of wild grasses, and management is difficult.

Plant Disease Profiles: Take-all root rot

Root rots

- caused mostly by fungi and oomycetes, but can also be caused by bacteria or nematodes

Armillaria Root Disease

Armillaria spp. cause root rots of woody plants. These basidiomycetes are identified by the clumps of characteristic honey-colored **mushrooms** produced at the base of infected trees, white mycelial fans beneath the bark, and black, cordlike **rhizomorphs** that allow the fungi to create a vast mycelial network from tree to tree (Figure 8.31). *Armillaria* was named the “humongous fungus” when it was discovered that the mycelium of a single genetic strain can cover many acres of a forest. It is a pathogen of conifers, hardwoods, and fruit trees. Once the roots are weakened by decay, the trees can be blown over in windstorms (see Chapter 2, Disease Classic: Armillaria Root Disease).

Figure 8.31. Rhizomorphs (“shoestrings”) of an *Armillaria* sp. adhering to roots of an apple tree.

Black Root Rot

Black root rot is caused by *Thielaviopsis basicola*, a soil inhabitant with a wide host range that includes many flower, ornamental, and vegetable plants. The fungus is identified by the production of tremendous numbers of thin-walled endoconidia, important for dispersal, and dark, thick-walled **chlamydospores** that function as survival structures (Figure 8.32). A simple trapping procedure with carrots can be used to determine whether the soil is contaminated by this pathogen. If carrot slices are buried in a moist soil sample for several days and then transferred to a moist chamber, the diagnostic spore stages will develop within a few days if *T. basicola* is present in the soil.

Figure 8.32. Chlamydospores (left) and endoconidia (right) of *Thielaviopsis basicola*, the black root rot pathogen.

Ectotrophic Root-Infecting Fungi

Ectotrophic, or runner, hyphae are produced by certain root-infecting ascomycete pathogens of grasses and cereal crops. Brown, septate hyphae grow along the root surface parallel to the roots and can be observed with a microscope (Figure 8.33). These ascomycetes live on the roots of host plants and cause infections in response to signals produced by root exudates. Disease examples include **take-all** of wheat (*Gaeumannomyces graminis* var. *tritici*) and bentgrass on golf courses (Figure 2.3) (*G. graminis* var. *avenae*), **necrotic ring spot** (*Ophiosphaerella korrae*) and **summer patch** (*Magnaporthe poae*) of bluegrass and fine fescue, and **spring dead spot** of Bermudagrass (caused by several *Ophiosphaerella* species).

Figure 8.33. Ectotrophic root-infecting hyphae (arrow), or runner hyphae, on a root of a turfgrass plant.

Fusarium Diseases

Fusarium spp. are ascomycetes that are often pink to yellow in color. Some species cause dry, reddish lesions on roots and crowns of herbaceous plants (Figure 8.34A). *Fusarium* spp. are easily identified by the spindle-shaped **macroconidia** (Figure 8.34B). Thick-walled **chlamydospores** aid in their long-term survival as soilborne pathogens.

Figure 8.34. A, Fusarium root rot of bean. B, Macroconidia of a *Fusarium* sp., showing the fusoid shape that gives the fungus its name.

Pythium and *Phytophthora* Root Diseases

In wet conditions, *Pythium* spp. and *Phytophthora* spp. are common root and crown oomycete pathogens. Many of these pathogens are soil inhabitants. **Oospores** aid in their long-term survival in soil (Figure 2.14). These soilborne organisms produce **zoospores** that are chemotactic and attracted to root exudates. In herbaceous plants, oomycetes often cause dark, soft rot of roots. Aboveground plant tissues near the soil surface may be infected when water carrying oomycete pathogens splashes up from the soil. Plant roots become more susceptible to infection in the low-oxygen conditions found in poorly drained soils because root growth and natural defense responses are reduced.

Rhizoctonia Diseases

Rhizoctonia spp. are basidiomycetes that cause reddish brown root lesions on a wide variety of herbaceous plants. They also invade plant parts in direct contact with the soil. For example, the lower surface of cucumbers lying on soil may develop **belly rot** from *Rhizoctonia* infection. Although *Rhizoctonia* spp. are important root pathogens of many kinds of plants, they may cause foliar diseases in turfgrass: **brown patch** (in hot weather) and **yellow patch** (in cool weather). *Rhizoctonia* spp. are soil inhabitants that produce numerous tiny reddish brown to black **sclerotia** as survival structures (Figure 8.35A). These species are identified by the characteristic **right-angled branching** of their hyphae (Figure 8.35B).

> **► DID YOU KNOW?**
>
> *Rhizoctonia* sclerotia are commonly found on potato tubers sold in supermarkets. The official name of the disease is black scurf. Most tubers have a few sclerotia, which some people call "the dirt that won't wash off."

Figure 8.35. A, Sclerotia of *Rhizoctonia* on a potato tuber with black scurf. B, Characteristic right-angled branching of *Rhizoctonia* hyphae.

Southern Blight

Southern blight is caused by the basidiomycete *Sclerotium rolfsii*, a soil inhabitant with a wide host range. It produces abundant white mycelium and numerous brown **sclerotia** (Figure 2.1). *S. rolfsii* can potentially attack any part of many plants. It is primarily a stem and crown pathogen, although the fungus may spread to the roots. The sclerotia are survival structures, but they are not cold tolerant, so the disease is primarily a problem in warm and tropical areas of the world. When infected transplants are shipped to greenhouses or to areas with cold winters, they can serve as sources of inoculum even though the pathogen will not survive exposure to winter conditions.

Root Rots

Plant Disease Profiles:

- **Aphanomyces root rot or common root rot of legumes**
- **Armillaria root disease** (see Chapter 2, Disease Classic)
- **Black shank of tobacco**
- **Brown root rot (tropical)**
- **Phytophthora stem and root rot of soybean**
- **Southern blight** (see Chapter 9, Disease Classic)
- **Sudden death syndrome of soybean (SDS)**
- **Take-all root rot** (see Chapter 8, Disease Classic)

Root rots are usually not noticed until considerable damage has already occurred, and the aboveground part of the plant begins to show symptoms. In most cases, laboratory examination of the roots and isolation of the pathogen are necessary for diagnosis. Most root rots are caused by fungi and oomycetes. Management of root diseases is difficult.

What is wood decay?

Decay of wood is caused primarily by basidiomycetes that produce relatively large fruiting bodies that may be shaped like mushrooms or conks (Figure 8.36). However, large basidiomycete fruiting bodies on or near trees are not necessarily indicative of wood decay. Many mushrooms that appear under trees are from beneficial ectomycorrhizal fungi that live in association with the tree roots (see Chapter 2). Saprophytic species also may produce fruiting bodies on dead branches but may not be a threat to the rest of the tree. Unlike canker pathogens, wood decay pathogens actually invade and destroy the secondary xylem (wood). Some of the decay pathogens cause **white rot**. These pathogens may utilize all components of wood, including cellulose, hemicellulose, and lignin, leaving the wood light-colored and spongy. Other wood decay pathogens cause **brown rot** by degrading primarily cellulose and leaving the lignin behind, so the wood becomes weak and crumbly.

Wood decay

- caused mostly by basidiomycetes
- pathogen enters plants through wounds
- spread by contact between roots
- spread by basidiospores

Figure 8.36. Fruiting body (conk) of a wood decay basidiomycete on the trunk of an infected tree. Basidiospores are produced in the tiny pores on the lower surface. Removal of the conk will not save the tree.

As with cankers, the size, number, and location of wounds play a role in disease severity. Even a small wound on the main trunk of a tree can serve as an invasion point for wood decay pathogens. Many trees are able to “compartmentalize” decay with a series of biological defense mechanisms (see Chapter 9). Stress factors such as drought, soil compaction, nutrient deficiency, and air pollution can reduce the ability of a tree to form defenses around wounds to help prevent infection.

Wood decay is caused by a group of specialized organisms, primarily basidiomycetes, that invade the secondary xylem (wood). Wounds provide an entry point for wood decay

fungi. Wood decay pathogens also may spread through root contact. There are no chemical treatments that can stop the decay.

What are postharvest diseases?

Humans often want to ship plant products to distant places or store them for future use. Cut flowers, fleshy fruits, tubers, bulbs, and roots may be infected or infested by storage pathogens before they are harvested, although the infections may be latent (not visible).

Many fruits become more susceptible to postharvest pathogens as they ripen (Figure 8.37). In some cases, fruits are dipped or washed in chemicals that disinfest the surface or in fungicides that reduce the growth of storage fungi. Produce is generally stored in cool conditions to reduce growth of postharvest bacteria and fungi. Storage facilities must provide a level of humidity that will minimize moisture loss from these stored, fleshy plant parts but will avoid condensation and formation of water droplets. Water droplets passing over stored products can easily disperse fungal spores and bacteria. Prevention of wounding during harvest and storage reduces infections.

DISEASE CLASSIC

Brown Rot of Stone Fruits

Hosts stone fruits (cherry, nectarine, peach, plum), almond, and occasionally some pome fruits (apple, pear)

Pathogens *Monilinia fructicola* and *M. laxa* (ascomycetes)

Symptoms and Signs Common symptoms are blossom and twig blight and soft rot of fruits, both on the trees and in storage after harvest. The brownish gray conidia quickly cover large round areas of the surface of infected fruits. Infected fruits left on trees may become mummified (dry rotted).

Disease Cycle Ascospores are forcibly ejected from asci in apothecia on fallen fruits. The more important primary inoculum comes from conidia produced on mycelia in small twig cankers, mummies on trees, and infected fruit stems.

Management Infected fruits and twig cankers should be removed. Fungicide protection is important in humid areas at flowering and again as the fruits mature. Harvested fruits should be handled carefully to prevent wounds and kept at low temperatures to reduce fruit rot.

Significance Brown rot is the most important disease of stone fruits in warm, wet climates worldwide.

Plant Disease Profiles: Brown rot of stone fruits

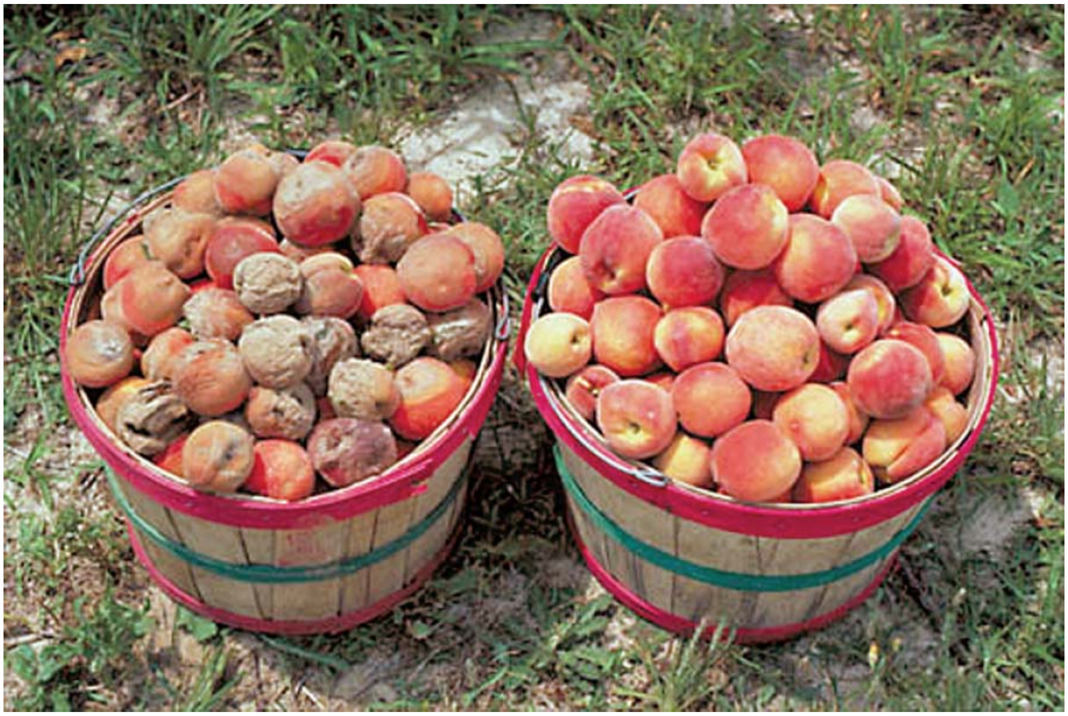


Figure 8.37. Peaches with brown rot (left), caused by *Monilinia fructicola.*

Stored seeds may be infested or infected by pathogens, and this contamination also often occurs before harvest. Storage facilities must be kept cool and dry to reduce fungal growth. Pathogens often enter seeds through feeding wounds caused by insects and rodents, so control of these pests may be required.

Some fungi (e.g., *Aspergillus, Fusarium*, and *Penicillium*) produce **mycotoxins** that can be very toxic to humans and animals at low concentrations (Figure 8.38). Even small amounts of moldy grain can result in significant mycotoxin contamination when the grain is processed (Figure 8.39). Mycotoxins are most common in cereal grains, corn, peanut, and tree nuts. Legal limits have been established for various mycotoxins in raw and processed products because mycotoxins usually cannot be detected by sight, smell, or taste in processed flour and other food products. Food safety laboratory tests are routinely conducted to monitor for mycotoxin contamination that exceeds legal limits (see Chapter 10 for further information).

► DID YOU KNOW?

Fusarium head blight (FHB) became a severe threat to wheat crops in the 1990s as a result of wetter-than-normal weather and the adoption of reduced tillage methods, which left Fusarium-infested stubble on the surface of the soil. Not only does the disease cause yield losses, but grain infected by *Fusarium* spp. may be contaminated by mycotoxins such as deoxynivalenol (DON), also known as vomitoxin. The U.S. Department of Agriculture (USDA) ranks FHB as the worst disease of wheat in the United States since the rust epidemics of the 1950s. Since 1990, wheat and barley farmers in the United States have lost over $3 billion due to FHB epidemics. In 1995, one company had to recall 16,000 tons of dog food because of vomitoxin contamination and fielded over 11,000 phone calls from unhappy pet owners (Figure 8.38).

Pathogens responsible for postharvest diseases of fleshy plant parts and seeds (grains) are usually present at the time of harvest. Growth of the pathogens is reduced by avoiding wounds and excess moisture, by providing cool storage temperatures, and occasionally by applying biological or chemical treatments. Postharvest fungi may produce harmful mycotoxins in food. Moldy fruits and vegetables are not usually consumed; however, contaminated grain and seeds sometimes are used in animal feed or processed foods such as flour. Mycotoxins in stored grains and seeds are an important public health concern.

PLEASE, NO
VOMITOXIN

Figure 8.38. Humans and other animals can be poisoned by mycotoxins, such as vomitoxin.

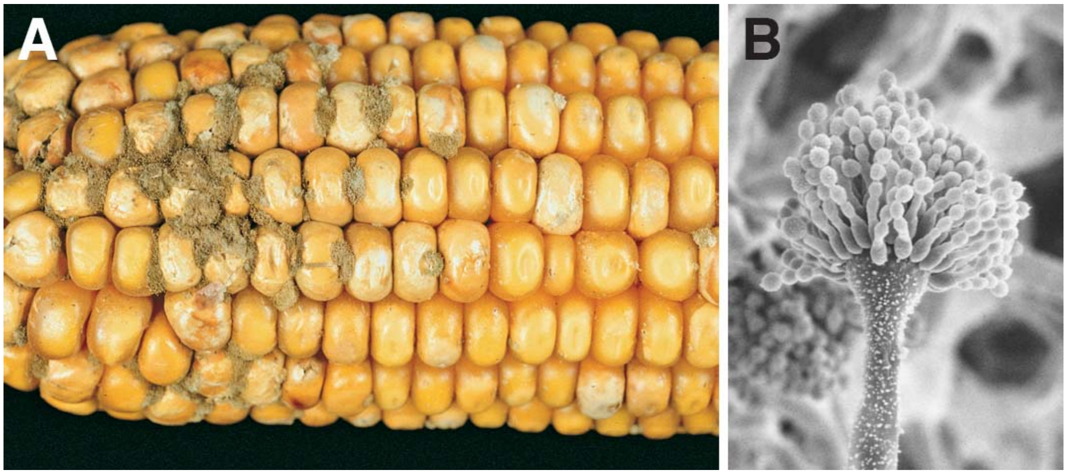


Figure 8.39. A, Aspergillus ear rot of corn. *Aspergillus flavus*, one fungal species that causes corn ear rot, produces aflatoxin, a dangerous mycotoxin. B, Conidiophore of *Aspergillus flavus*. Note the numerous conidia (scanning electron micrograph, × 1,000).

► DID YOU KNOW?

Aspergillus flavus produces **aflatoxin**, a dangerous carcinogenic mycotoxin. *Aspergillus* is named for the arrangement of the conidia and conidiophore, which resembles the shape of an aspergillum, a device used by Catholic priests to sprinkle holy water. Like *Penicillium, Aspergillus* is an ascomycete. It is common in corn and high-oil seeds, such as cottonseed (which is fed to animals), peanut, and tree nuts. The legal limit of aflatoxin in food in the United States is 20 parts per billion. In 2005, hundreds of dogs were poisoned in the eastern United States because aflatoxin-contaminated corn was accidentally used in commercial dog food.

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Questions

1. What is damping-off, and why is it different from most diseases of mature plants?
2. What symptoms and signs could help you differentiate a leaf spot disease caused by a bacterium from a leaf spot disease caused by a fungus?
3. What are four foliar diseases that can usually be easily diagnosed by observation of abundant fungal structures? How is each identified?
4. What fungal structures are characteristic of most anthracnose diseases? How does the management of the host plant impact the management of anthracnose as a disease? Consider, for example, anthracnose of sycamore compared to anthracnose of turfrass.
5. What information do you need to determine whether removal of an alternate host is a viable management option for a heteroecious rust?

6. The barberry eradication program did not eliminate wheat stem rust from North America, but it was still considered a success. What are some benefits that did result from the program? Why are cereal rust specialists concerned about the regrowth of wild barberry plants?
7. What part of the plant is affected by vascular wilt? What are some important differences between Verticillium wilt and Fusarium wilt? How could you determine which pathogen was present without sophisticated laboratory equipment?
8. Some virus infections commonly begin from leaf inoculations, but other viruses are restricted to the vascular system. How do the viruses that cause these two types of diseases differ in the host tissues that they infect? In the ways they are transmitted?
9. What are important differences between a bacterial vascular wilt such as Stewart's wilt of corn and a wilt disease caused by a xylem-limited fastidious bacterium such as *Xylella fastidiosa*?
10. What part of the plant is affected by a canker? Name two important canker diseases. How are canker diseases different from wood decay? What factors favor both types of diseases in woody plants?
11. Which kinds of pathogens can cause galls in plants?
12. Why are root diseases difficult to diagnose and manage?
13. What is a mycotoxin? Why are mycotoxins of more concern in stored grains and seeds than in fresh fruits and vegetables that rot after harvest?

Challenge Questions

1. Why is rust usually a minor problem on turfgrasses but a significant disease of wheat and other cereals? How can rust affect the price of turfgrass seed?
2. Your neighbor wants you to remove your pine tree that was recently diagnosed with white pine blister rust (*Cronartium*

ribicola) to protect the pines in his yard. Is this a reasonable request?

3. An elm tree may have Dutch elm disease or Verticillium wilt. How could you determine which disease it has? How would the diagnosis affect your management choices?
4. Recently a *forma specialis* of *Fusarium oxysporum* was discovered that infects coca, the plant from which cocaine is derived. Drug enforcement agents have been using herbicides to kill coca plants, but they wonder if they should try this biological approach to kill the plants. What would be the advantages of using Fusarium wilt instead of herbicides to kill coca plants? How should the fungus be applied to most effectively cause disease? Is this biological control or biological warfare?
5. Discuss the role of insects in the transmission of plant pathogens. Consider all pathogen groups that are transmitted and the specificity of the transmission relationship, i.e., is the pathogen spread in a general way by insects or only by certain insects feeding in specific plant tissues?
6. Many insects, mites, and related organisms cause galls on plants. Should these organisms be considered pathogens? Are these gall diseases?

Supplementary Resources

Illustrated Glossary

Special Topic

Mycotoxins in crops: A threat to human and domestic animal health

Plant Disease Profiles

Leaf Spots, Scab, and Blight Diseases

- Anthracnose of turfgrass
- Apple scab
- Bacterial leaf scorch
- Bacterial spot of pepper and tomato
- Black Sigatoka of banana and plantain
  - Sigatoka negra da bananeira e platanos (Portuguese)
  - Sigatoka negra de bananeros y plátaneros (Spanish)
- Dollar spot of turfgrass
- Early blight of potato and tomato
  - Pinta-preta (Alternariose) da batata e tomate (Portuguese)
- Fire blight
- Fusarium head blight
  - Giberela ou Fusariose (Portuguese)
- Late blight of potato and tomato
  - 马铃薯和番茄晚疫病 (Chinese)
  - Requeima (mildio, pt) da batateira e tomateiro (Portuguese)
- Phytophthora blight of curcurbits
- Pythium blight of turfgrass
  - Quemazón del césped por Pythium (Spanish)
- Rhizoctonia diseases of turfgrass
- Rice blast
- Sudden oak death, ramorum leaf blight, ramorum shoot blight
- Taro leaf blight
- White mold (Sclerotinia)

Downy Mildew

- Downy mildew of grape

Rusts

- Coffee rust
- Soybean rust
  - Ferrugem asiática da soja, Ferrugem da soja. (Portuguese)

- Stem rust of wheat
- White pine blister rust

Smuts

- Common smut of corn
- Stinking smut of wheat

Vascular Diseases

- Bacterial leaf scorch
- Barley yellow dwarf
- Dutch elm disease
- Fusarium wilt of watermelon and other cucurbits
- Lethal yellowing of coconut
- Pine wilt (nematode)
- Stewart’s wilt of corn
- Verticillium wilt

Virus and Viroid Diseases

- Barley yellow dwarf
- Cucumber mosaic
- Soil-borne wheat mosaic
- Spindle tuber disease
- Tobacco mosaic
- Tomato spotted wilt

Cankers

- Citrus canker
- Dwarf mistletoes
- Fire blight of apple and pear
- Leucostoma canker of stone fruits
- Sudden oak death, ramorum leaf blight, ramorum shoot blight

Galls

- Black knot
- Common smut of corn
- Crown gall

- Root-knot nematodes

Root and Stem Diseases

- Aphanomyces root rot or common rot of legumes
- Armillaria root disease
- Black leg of oilseed rape, including canola
- Black leg of potato
- Black shank of tobacco
- Brown root rot
- Burrowing nematode
- Lesion nematode
- Monosporascus root rot and vine decline of melons
- Phytophthora root and stem rot of soybean
- Southern blight
- Soybean cyst nematode
- Sting nematode
- Sudden death syndrome of soybean (SDS)
  - Síndrome de la muerte súbita (SMS) de la soja (Spanish)
- Take-all root rot
- White mold (Sclerotinia)

Postharvest Disease

- Brown rot of stone fruits

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

- acervulus (pl. acervuli)
- aecium (pl. aecia)
- alternate host
- anthracnose
- ascospore

ascus (pl. asci)
autoecious
avoidance
basidiospore
biotroph
blight
blotch
brown rot (of wood)
canker
chasmothecium (chasmothecia)
chemotaxis
chlamydospore
cleistothecium (pl. cleistothecia)
conidium (pl. conidia)
damping-off
demicyclic
dikaryotic
downy mildew
ectotrophic
endoconidium (pl. endoconidia)
fastidious
flag
flower break
forma specialis (pl. *formae speciales*)
gall
girdle
haustorium (pl. haustoria)
heteroecious
hyperplasia
hypertrophy
leaf spot
macroconidium (pl. macroconidia)
macrocyclic

microconidium (pl. microconidia)
microcyclic
microsclerotium (pl. microsclerotia)
mosaic
mottle
mycotoxin
necrosis (adj. necrotic)
needle cast
obligate parasite
oospore
perithecium (pl. perithecia)
phytoplasma
plasmid
powdery mildew
Puccinia pathway
rhizomorph
ringspot
rugose
rust
scab
sclerotium (pl. sclerotia)
seta (pl. setae)
smut
spiroplasma
sporangiophore
sporangium (pl. sporangia)
sporidium (pl. sporidia)
sporodochium (pl. sporodochia)
synnema (pl. synnemata)
teliospore
telium (pl. telia)
urediniospore
uredinium (pl. uredinia)

vascular wilt
vein banding
white rot (of wood)
white rust
xylem-limited fastidious bacteria
zoospore

Exercises

Exercise 8.1. Using the APS list *Common Names of Plant Diseases*, choose a host plant and make a list of the common diseases described in this chapter that affect that plant.

Exercises 8.2 and 8.3. Identify the diseases in the images presented.

Internet Research

Internet Research Exercise 8.1. Consult a list of diseases of a plant of interest. Choose an example of each of the following diseases:

leaf spot
root rot
anthracnose
rust
virus

For the leaf spot disease

a. What are the symptoms?
b. What is the name of the pathogen? Is the pathogen fungal or bacterial?
c. Is this disease a high-nitrogen or a low-nitrogen problem, i.e., is disease more severe when foliage is lush or when it is nitrogen-deficient?

d. What signs can be used for diagnosis (bacterial streaming, fungal spores, fruiting bodies)?
e. What management practices are recommended?
f. Is fungicide or other chemical protection required or recommended?

For the root rot disease

a. What are the diagnostic symptoms and signs of the disease?
b. What is the name of the pathogen?
c. What management practices are recommended?
d. If fungicide is recommended, how should it be applied and what results can be expected?

For the anthracnose disease

a. What are the symptoms and signs of the disease?
b. What is the name of the pathogen?
c. What parts of the plant are most severely affected?
d. What management practices are recommended?

For the rust disease

a. Is the rust fungus autoecious or heteroecious?
b. What is the name of the pathogen?
c. What are the hosts?
d. If there is a urediniospore stage (the repeating stage), on which host is it found?
e. If the rust fungus is heteroecious, is removal of one host an economically viable management approach? Which host would you remove and what impact would this have on the disease? Would additional management be required?
f. If the rust fungus is autoecious, what management practices are recommended?

For the virus disease

a. What are the symptoms of this virus disease?
b. What is the name of the virus?
c. Does the virus have any vectors? If not, how is it spread?

d. Are resistant cultivars available?
e. What other management practices are recommended?

Chapter 9

How Do Plants Interact with Pathogens?

IN THIS CHAPTER:

Part A. Ecological interactions

- ► **How do pathogens survive in the absence of host plants?**
- ► **How do pathogens find plants?**
- ► **How do pathogens penetrate plants?**

Part B. Physiological interactions

- How do pathogens establish infections?
- How do plants defend themselves?

Part C. Genetic interactions

- **What are the genetic interactions between plants and pathogens?**
- **How can we create disease-resistant plants?**

Plants exist in a world filled with microorganisms (Figures 9.1 and 9.2). They continue to grow in the same location throughout a growing season or for many years, as in the case of perennial plants. Their surfaces are constantly exposed to bacteria, fungi, nematodes, and possibly parasitic plants. They may be inoculated with viruses during feeding by insects or by other vectors.

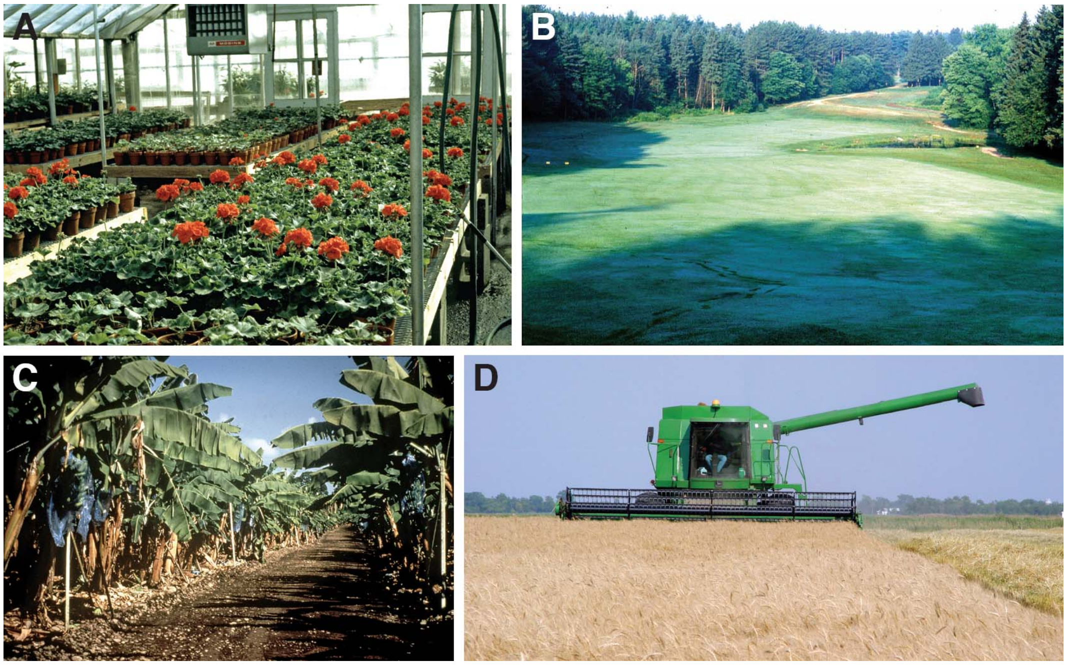


Figure 9.1. Healthy plants in various agroecosystems. Potential pathogens are not visible but are certainly present. A, Geranium crop in a greenhouse. B, Turfgrass on a golf course fairway. C, Bananas on a plantation in Puerto Rico. D, Wheat harvest in the United States.

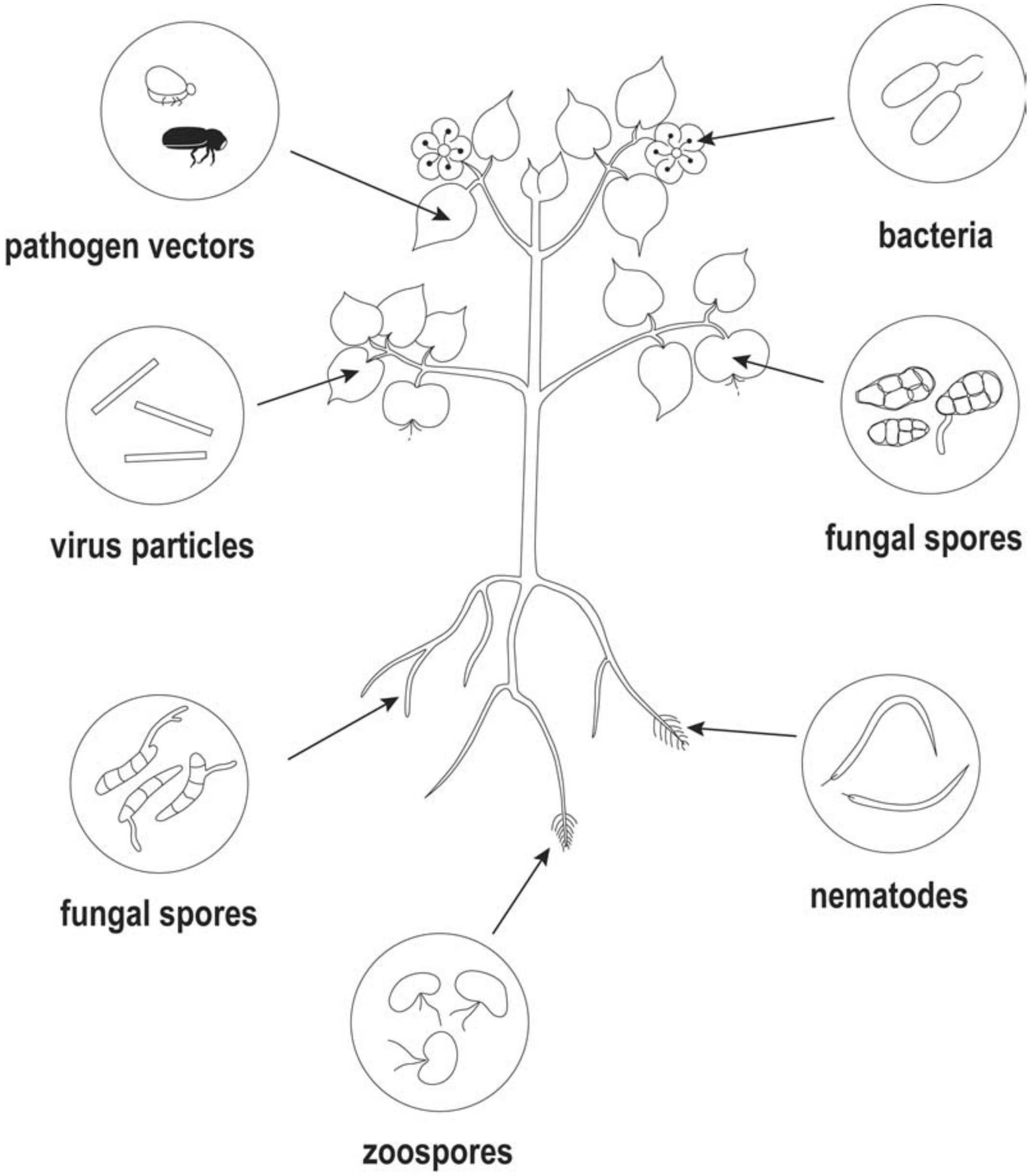

pathogen vectors
bacteria
virus particles
fungal spores
fungal spores
nematodes
zoospores

Figure 9.2. Plants are exposed to a variety of pathogens.

Most plants are immune to most plant diseases. Of the multitude of bacteria, fungi, nematodes, and viruses, only some are capable of establishing a parasitic relationship with a plant. Of these, only a small subset can infect a particular plant species. Therefore, the study of plant diseases really focuses on a limited group of potential pathogens. Even though the complete list of pathogens for a plant species may appear quite long, it includes many minor pathogens. If you choose a specific plant in a specific environment, it is possible to become familiar with all of the likely pathogens and then focus on the diseases that have significant economic impact on yield and/or quality.

Many of the microorganisms present in a plant's environment are harmless, and some are even beneficial. For example, various bacteria and fungi colonize plant surfaces (including leaves, fruits, and especially roots) and live on exudates from plant cells without infecting the plant or causing disease. The natural competition among these organisms affects pathogen populations and can be exploited for biological control.

Plant pathogens have made many adaptations to enable them to invade plants, overcome plant defense mechanisms, and colonize plant tissues for growth, survival, and reproduction. Once established inside the plant, they have at least temporarily escaped the intense competition from saprophytic organisms on plant surfaces and in the soil.

Plants respond to the threat of invasion by pathogens with many defenses, some of which are always present (**constitutive** or **passive defenses**) and others that are triggered in response to invasion (**induced** or **active defenses**). The constitutive defenses help to keep most potential pathogens at bay. The plant also must be able to detect the presence of an invading pathogen so that it can fight back with its induced defenses. Plant defenses, both constitutive and induced, may be structural or chemical and are an important component of genetic resistance to disease.

Most of the biotic pathogens of plants are parasites. Parasites are an important part of every ecosystem, and host-parasite interactions are of interest to plant ecologists. Recognizing the amazing interactions between plants and their parasites is fundamental to an understanding of agricultural ecosystems as well. This understanding helps us to modify our cultural practices in ways that benefit the plant and/or negatively impact the pathogens. The bases of many recommended cultural practices are more easily understood in light of host-parasite interactions.

The way in which we grow any given plant influences whether a plant pathogen will cause disease and become economically significant or whether disease will be successfully avoided. In this chapter, we focus on three important kinds of biological interactions between pathogens and their host plants: ecological, physiological, and genetic interactions.

Part A: Ecological Interactions

IN THIS SECTION:

How do pathogens survive in the absence of host plants?

- Survival structures
- Survival in perennial plants and seeds
- Survival in vectors
- Survival in host plant debris
- Survival in soil
- Survival as epiphytes

How do pathogens find plants?

- Above ground
- Below ground

How do pathogens penetrate plants?

How do pathogens survive in the absence of host plants?

If one views a plant from the perspective of a potential pathogen, some interesting problems present themselves. Perennial plants such as trees and turfgrasses appear to be stable homes for pathogens, but many plants are annuals and may not be present at all times. Foliar pathogens of deciduous trees and perennial herbaceous plants may not find available plant tissue during winter or dry seasons. Sometimes a diseased plant dies and becomes colonized by competitive saprophytes and secondary invaders, so the pathogen must find a new host plant. In any of these cases, pathogens must have some means of survival in the absence of a host plant.

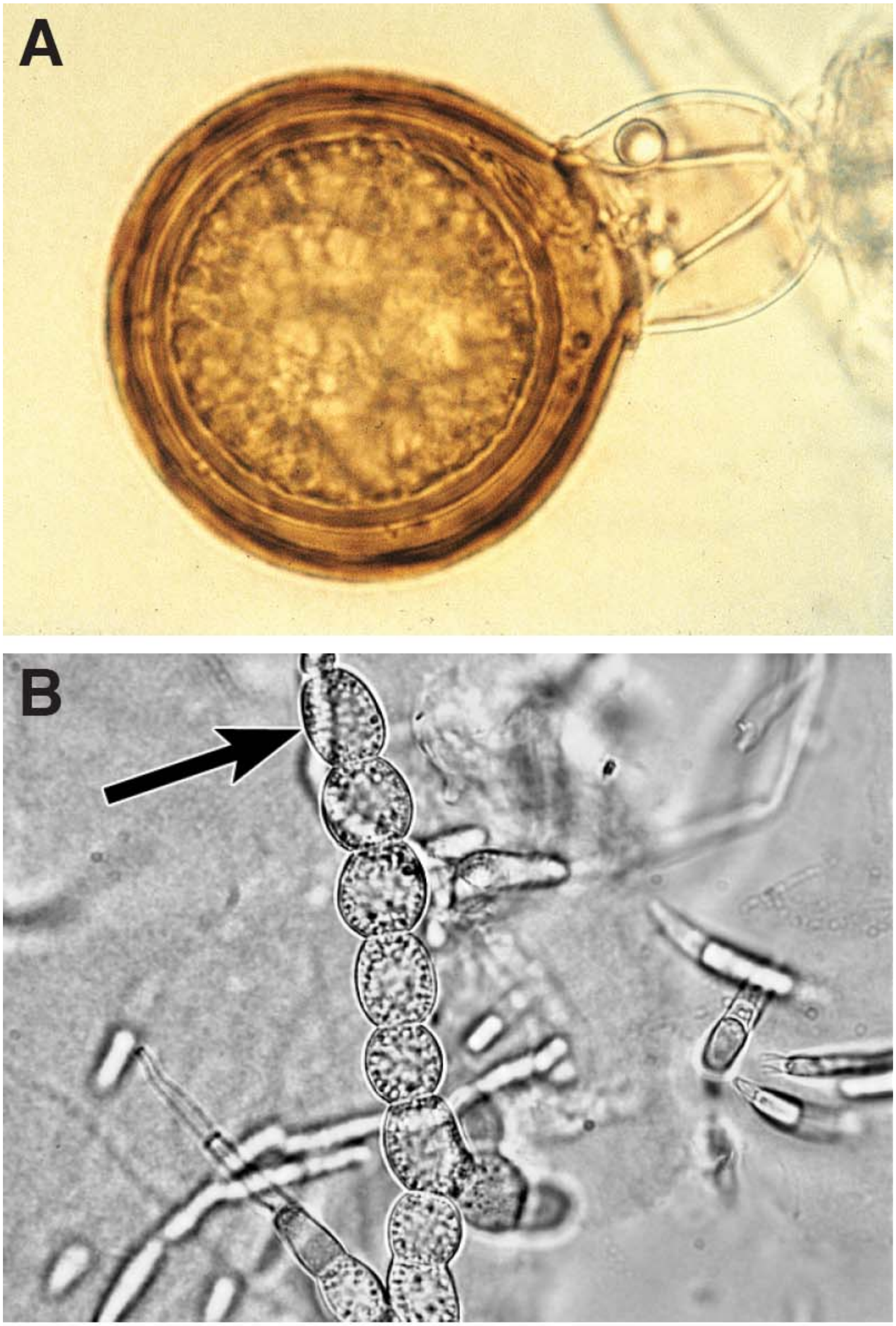
Plant pathogens have evolved numerous survival strategies. Sometimes the pathogen is in an inactive, or dormant, stage. In other cases, it remains active as a pathogen of other organisms or as a saprophyte.

Survival Structures of Fungi and Fungus-Like Organisms

Spores	Structures
chlamydospores	chasmothecia
oospores	perithecia
teliospores	pseudothecia
thick-walled conidia	pycnidia
zygospores	sclerotia

Survival Structures

Fungi and fungus-like organisms, in particular, produce a variety of survival structures that help them remain alive in plant debris and in soil. In some cases, the thick cell wall of a spore protects the living protoplasm inside from desiccation and ultraviolet (UV) radiation (Figure 9.3A and B). An even more effective protection results when fungi produce **melanized** cells, in which dark pigments are deposited in the cell wall. Melanin helps to protect against UV radiation and microbial enzymes that might degrade the fungal cell wall. Melanization can occur in the walls of individual spores or in the walls of cells that make up the outer layer of a fruiting body filled with spores (Figure 9.3C). In **sclerotia**, a melanized rind of cells is produced around a mass of living hyphae (Figure 2.19). Although not a spore or fruiting body, these melanized masses of hyphae serve as survival structures, or propagules. When sclerotia become active under conducive environmental conditions, new mycelia emerge from them.



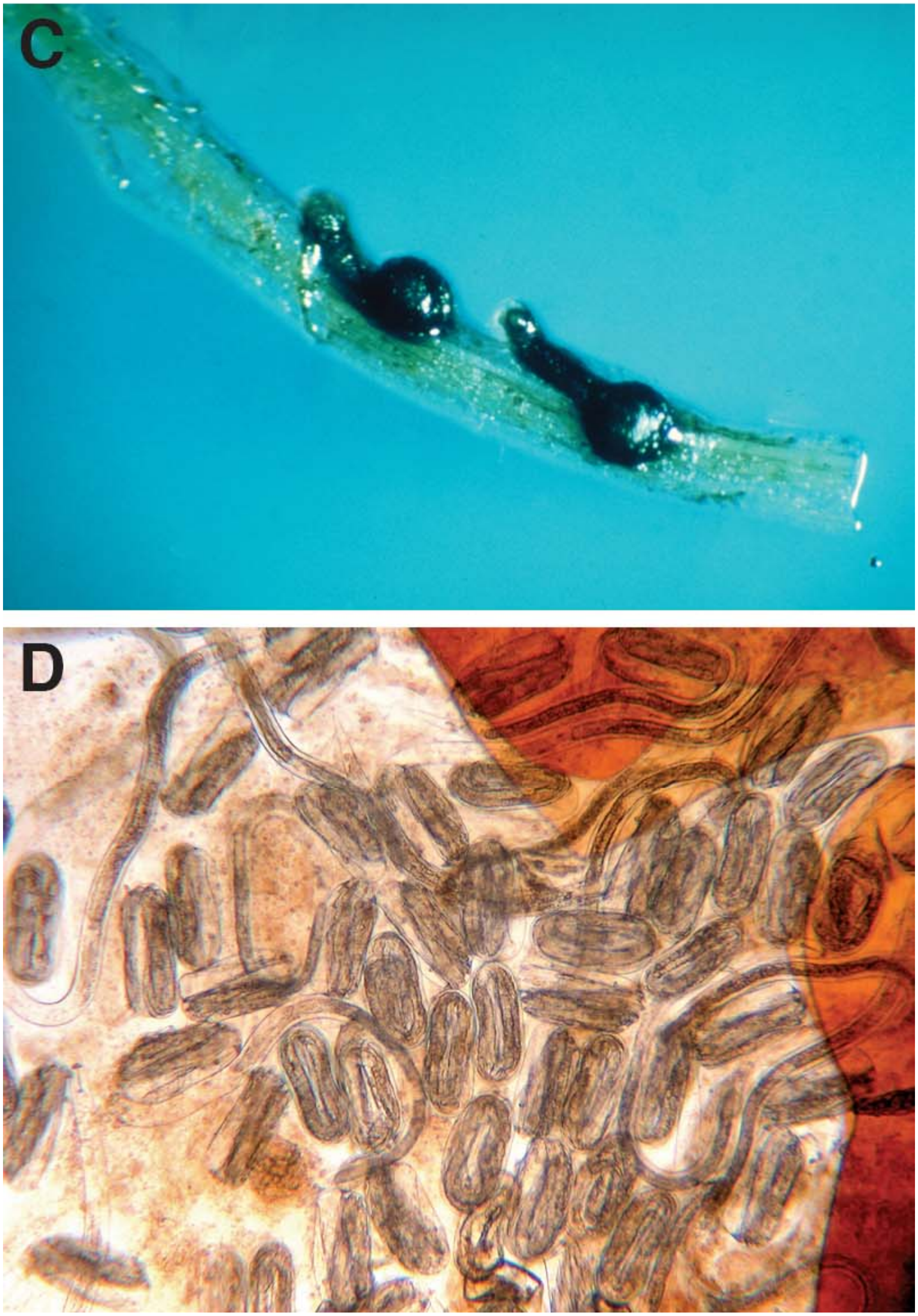


Figure 9.3. Examples of pathogen survival structures. A, Thick-walled oospore of *Phytophthora cinnamomi.* B, Chlamydospores (arrow) of *Fusarium sporotrichioides.* C, Melanized perithecia of *Gaeuman-nomyces graminis* var. *avenae* (take-all root rot). D, Nematode cyst opened to expose eggs and second-stage juveniles.

DISEASE CLASSIC

Southern Blight

Hosts more than 500 plant species, including apple, peanut, potato, tomato

Pathogen *Sclerotium rolfsii*. Teleomorph: *Athelia rolfsii* (basidiomycete)

Symptoms and Signs *S. rolfsii*, a soilborne pathogen, causes crown and stem rots, root decay, and soft rot of tubers and fleshy organs, and it sometimes attacks leaves of low-growing plants. Signs include mats of white mycelia and numerous small, brown sclerotia.

Disease Cycle Hyphae in plant debris and germinating sclerotia directly infect plant tissues near the soil and produce enzymes (oxalic acid, polygalacturonase, and cellulase) that contribute to the soft rot. Sometimes basidiospores are produced as secondary inoculum. They infect by producing an appressorium and a penetration peg. New sclerotia form about 5–7 days after infection. The fungus grows optimally in hot, humid conditions. It is generally killed by freezing temperatures. Sclerotia may survive a freeze, but will die if buried 15 centimeters (6 inches) deep for 45 days.

Management Infected plant materials should not be introduced to new sites. Soil may be treated with heat (e.g., solarization), chemicals, or biological agents. Nonhost crops should be used in rotations. Infested debris can be tilled into the soil to bury sclerotia. Infected plants and infested soil should be removed when possible.

Significance *S. rolfsii* is a very important and difficult-to-manage soilborne pathogen in humid tropical and subtropical areas.

Plant Disease Profiles: Southern blight

Most adult and juvenile stages of nematodes are vulnerable to dry or very cold conditions. Their populations typically decline during drought and in winter, but certain types of nematodes have specialized survival mechanisms. For example, some nematodes temporarily suspend metabolic activity under unfavorable environmental conditions. They can remain alive in this state (**cryptobiosis**) for years, returning to activity when conditions are more favorable. Although most nematode eggs are quite vulnerable, the eggs of root-knot nematodes are protected from desiccation by the gelatinous matrix in which they are laid. Cyst nematode eggs are protected within the female's body, which becomes a tough leathery sac, or cyst, after the adult female dies (Figure 9.3D).

The seeds of parasitic flowering plants are the survival structures of these pathogens. In contrast to such plants, as well as fungi and nematodes, most plant-pathogenic bacteria and viruses do not produce any specialized survival structures.

Fungi produce numerous kinds of survival structures that contribute to their success as plant pathogens. Nematodes may survive as eggs, especially in cysts, or by cryptobiosis. Parasitic flowering plants survive as seeds. Plant-pathogenic bacteria and viruses do not typically produce survival structures.

Survival in Perennial Plants and Seeds

Fungal mycelia or spores, bacteria, viruses, and some nematodes may be present in or on buds, bark, roots, and sometimes leaves of perennial plants. In the absence of an economically important host plant, a number of biotrophs (obligate parasites) survive in **alternate hosts** and **alternative hosts**, such as weeds or other perennial plants that remain alive even in the winter. Alternate hosts are the unrelated plant species required by a heteroecious rust fungus for the completion of its life cycle. Alternative hosts are plant species that may serve as hosts of a pathogen in addition to the economically valuable plants on which the pathogen lives. Alternative hosts are common survival sites for many nematodes, viruses, and fastidious bacteria such as phytoplasmas.

Humans aid pathogen survival by harvesting and storing seeds and other propagative parts and then planting them when conditions are favorable for both plant growth and pathogen growth. When the seed coat is contaminated with a pathogen but the cotyledons and embryo remain healthy, the seed is **infested**. The surfaces of infested seeds may be treated to reduce the contamination and help prevent infection of the germinating plants by bacteria, fungi, and viruses. **Infected** seed, in which the embryo or cotyledons are already colonized by the pathogen, is sometimes treated to eliminate a pathogen, but care must be taken to kill the pathogen without harming the plant embryo.

Any part of the plant that people save for new plantings (seeds, bulbs, corms, cuttings, rootstocks, and tubers) can serve as a survival site for pathogens. Pathogens may survive in active or inactive stages in these large masses of host tissue. For example, sclerotia of *Botrytis* spp. (gray mold), *Rhizoctonia* spp., *Sclerotinia sclerotiorum* (white mold), and *Sclerotium rolfsii* (Southern blight) often cling to underground propagative parts, such as bulbs, corms, rhizomes, and tubers, and are then planted along with them (Figure 9.4). Bacteria, nematodes, and viruses are more likely to be inside plant tissues. They are usually in an active state but may multiply more slowly in winter, in cold storage, or under drought conditions.

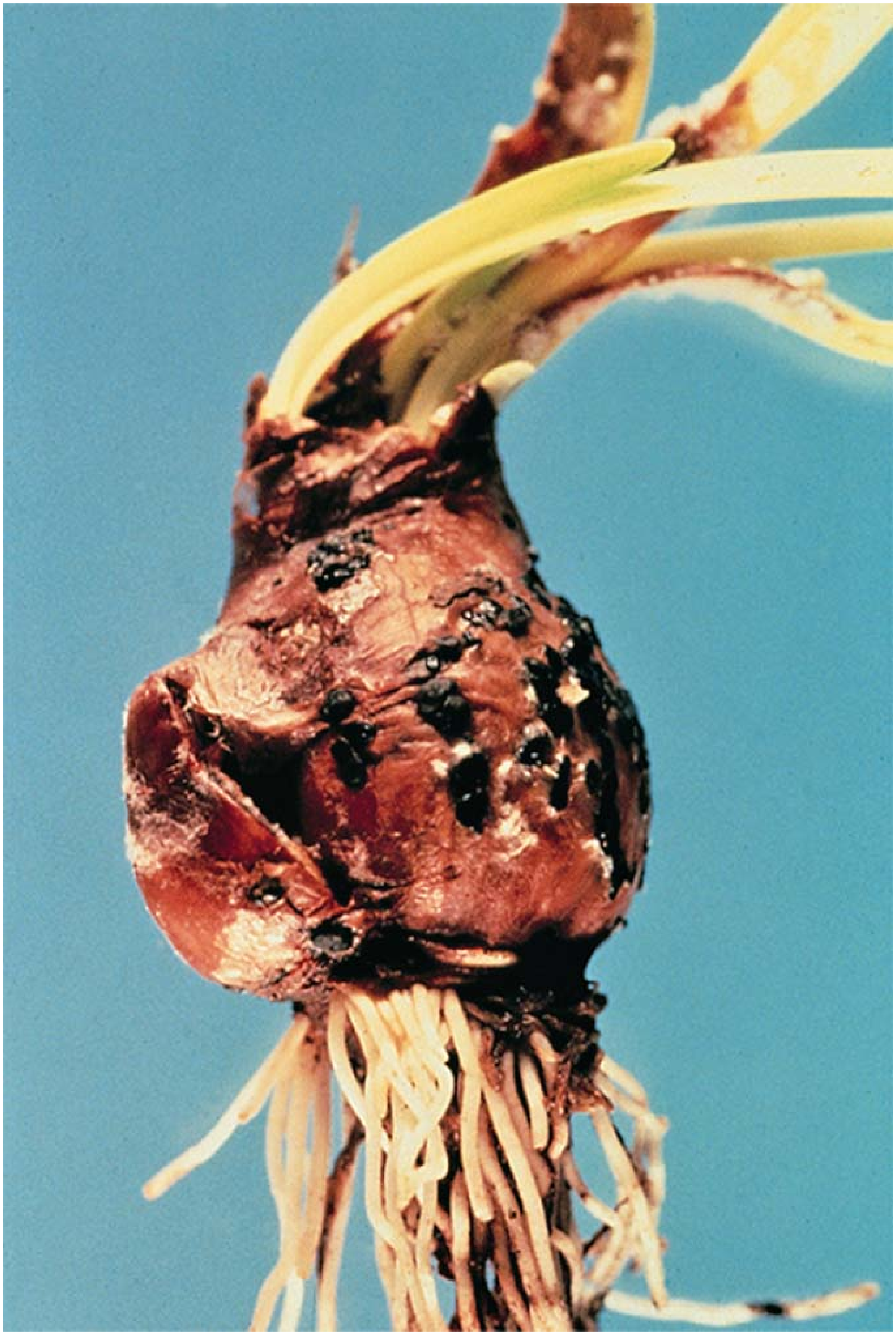


Figure 9.4. Sclerotia of *Botrytis* sp. (gray mold) and other fungal pathogens can contaminate bulbs and other propagative parts, leading to unintentional spread of the disease.

Perennial woody plants that are hosts of plant pathogens can be reservoirs of inoculum. Cankers in woody tissues are common overwintering sites for many bacterial and fungal pathogens (Figure 9.5). For example, fire blight bacteria (*Erwinia amylovora*) ooze out of cankers in the spring onto the surface of the plant, where they can be splashed or carried by insects to new host plants. The fungal pathogens that cause black knot of cherry, black spot of rose, chestnut blight, Cytospora canker of spruce, and various canker diseases of grape and bramble cane fruits (and many other pathogens) survive in cankers. After wetting by rain, these fungi produce large numbers of fruiting bodies that exude masses of spores onto the surface of the bark. Wood-decay fungi produce annual or perennial fruiting bodies that disperse airborne basidiospores capable of initiating new infections; spores are released over the several years that it may take a tree to die as it slowly rots away.

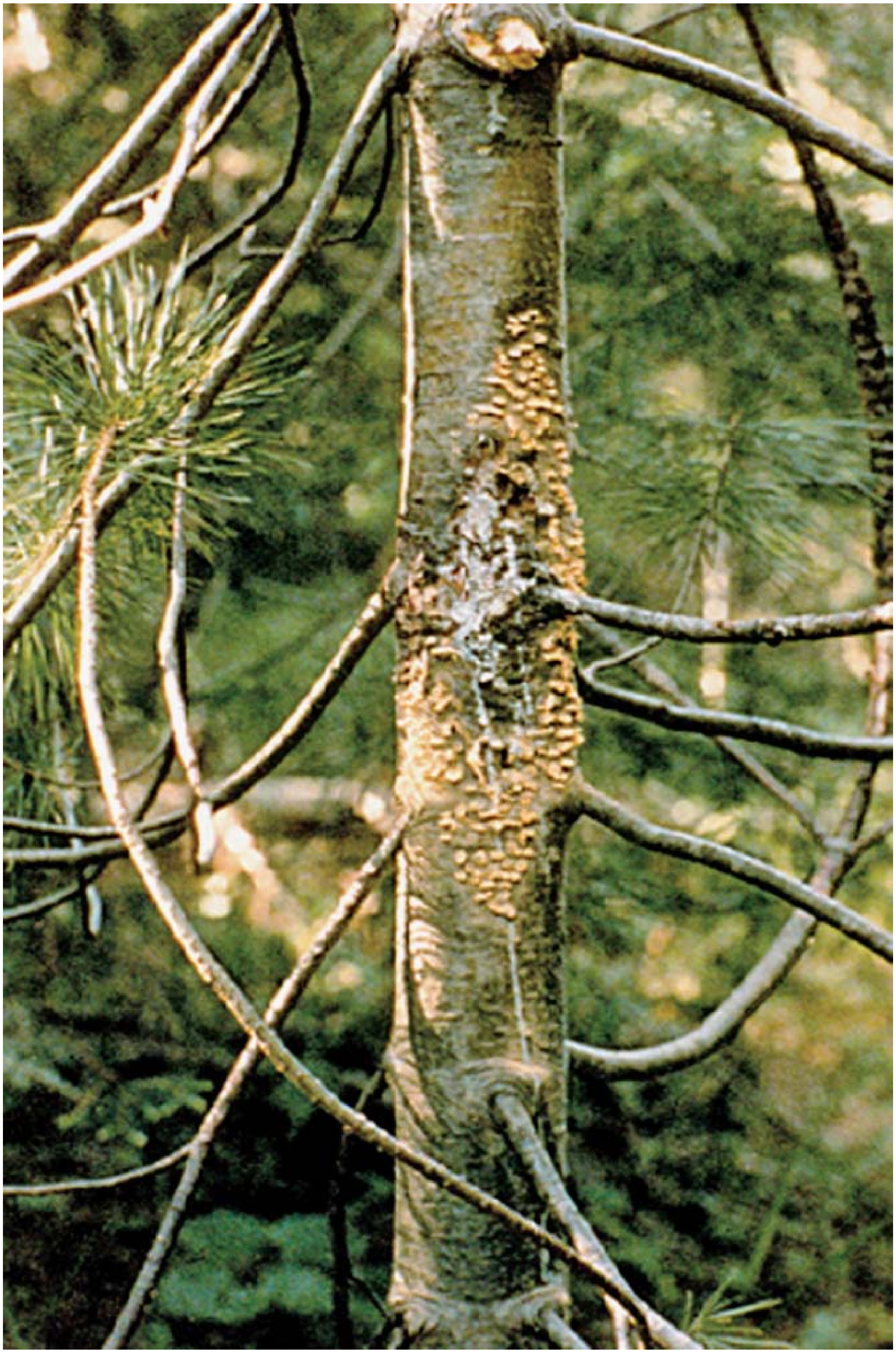


Figure 9.5. *Cronartium ribicola,* **which causes white pine blister rust, survives in blister rust cankers.**

Pathogen Survival Sites in or on Plants and Plant Parts

- cankers
- decaying wood
- propagative parts
    - bulbs
    - corms
    - cuttings
    - rhizomes
    - rootstocks
    - tubers
- perennial alternate hosts of heteroecious rust fungi
- perennial alternative hosts (including weeds)
- seeds

► **DID YOU KNOW?**

Living bacteria (*Corynebacterium agropyri*) were recovered from dried bacterial ooze on specimens of infected plants of a wild grass (*Agropyron smithii*) stored in a classroom at room temperature for 37 years. Although unlikely to occur in nature, this demonstrates the importance of ooze in the survival of plant-pathogenic bacteria.

Perennial plant parts (bark, buds of woody plants, roots, seeds, and tubers), cankers, and vectors are common survival sites for plant pathogens.

Survival in Vectors

Vectors of plant pathogens seek out shelter to survive winter conditions or the absence of actively growing host plants. These sheltered conditions also aid the survival of any plant pathogens that they carry, so the pathogens can be transmitted to a new plant when favorable conditions and host plants coincide. For example, the bacterium that causes Stewart's wilt of corn (*Erwinia stewartii*, syn. *Pantoea stewartii*) survives in the gut of overwintering corn flea beetles and is introduced into new plants as the insects feed in the spring in the midwestern United States. *Barley yellow dwarf virus* arrives in cereal fields each year carried by its aphid vectors. Elm bark beetles (Figure 9.6) are attracted to dying elm trees and overwinter under the bark. If the tree died of Dutch elm disease, the beetles are likely to emerge in the spring covered with the sticky spores of the *Ophiostoma* fungi that cause Dutch elm disease.

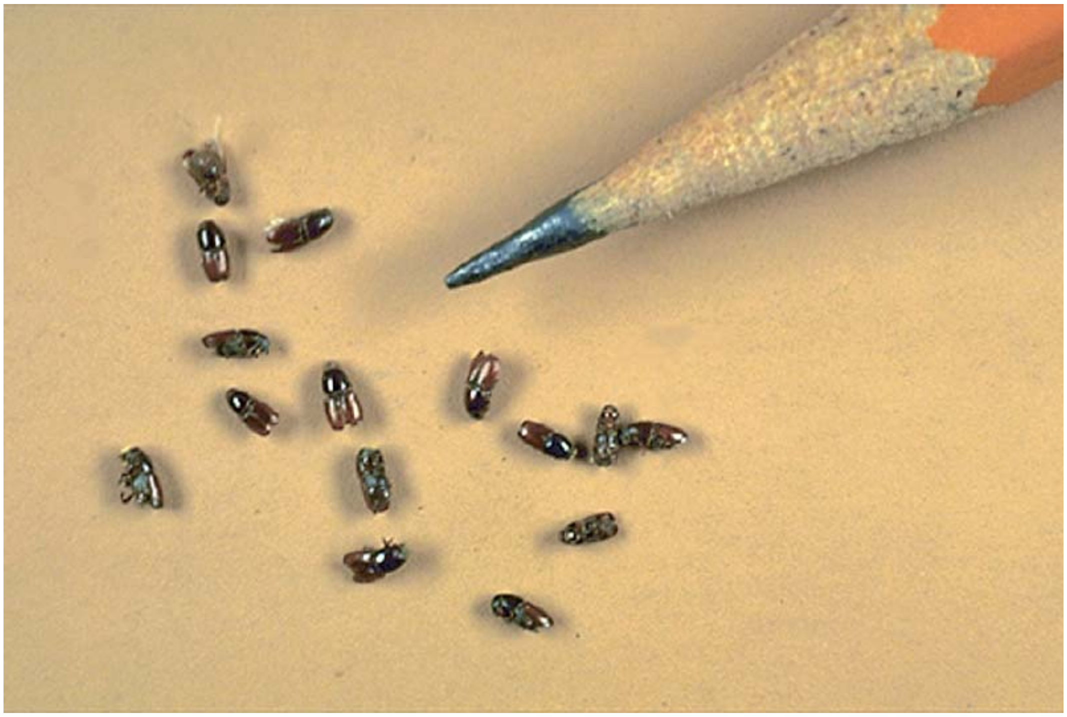


Figure 9.6. Elm bark beetles survive under the bark of dying elm trees, along with the fungal pathogens that cause Dutch elm disease.

DISEASE CLASSIC

Stewart’s Wilt of Corn

Host corn (maize)

Pathogen *Erwinia stewartii*, syn. *Pantoea stewartii*

Symptoms and Signs Symptoms include wilt and leaf blight on seedlings and long streaks of chlorosis or necrosis on infected leaves. Premature leaf death may make plants more susceptible to stalk rot. Bacterial streaming at the cut edge of an infected leaf can be observed under a microscope.

Disease Cycle The pathogen overwinters in the digestive tract of corn flea beetles and is introduced by fecal contamination of feeding wounds by the beetles; secondary cycles occur when new generations of beetles emerge. Disease risk can be predicted by monitoring winter temperatures that determine flea beetle survival. The pathogen may be seedborne.

Management Infected seed should be excluded from areas where the disease does not occur. Corn cultivars with genetic resistance are available. Plants are sometimes treated with insecticides to kill flea beetle vectors.

Significance Stewart’s wilt is an important disease in the midwestern United States. It caused significant losses before resistant hybrid field corn was developed. It is now a problem mostly in sweet corn that is susceptible but also has desirable agronomic attributes.

Plant Disease Profiles: Stewart’s wilt of corn

Some plant pathogens survive in vectors as vector pathogens. For example, **mollicutes** (phytoplasmas and spiroplasmas) survive in various leafhopper species. Because they are parasites of both leafhoppers and plants, they multiply in the vector, and therefore the vector remains capable of spreading the mollicute for its lifetime.

Survival in Host Plant Debris

Plant debris from a previous crop can be an important survival site for plant pathogens. Some common sources of plant debris harboring pathogens are stubble left behind after harvest of a field crop, fallen leaves from woody plants in landscapes and orchards, and thatch in turfgrasses. Biotrophs that cannot produce a survival structure will die when living plant cells are no longer available. Although some pathogens remain in dead plant tissues as survival structures, many pathogenic fungi and bacteria are facultative saprophytes or facultative parasites and are therefore capable of a saprophytic existence. For example, *Venturia inaequalis*, the fungus that causes apple scab, changes from a surface scab pathogen into a saprophyte that colonizes fallen apple and crabapple leaves. Such facultative organisms may face competition from secondary invaders and obligate saprophytes that also colonize the dead tissue.

Pathogens in dead host tissue are still relatively protected from the full onslaught of the microbial competition found in soil. When soil is tilled (plowed), however, plant debris is broken up and turned into the moist soil, where active microbial populations can more quickly degrade the organic matter and force pathogens into direct competition with the soil inhabitants (see below) (Figure 9.7). The removal of fallen leaves of trees and debris of annual plants may significantly reduce disease by removing the survival site and the source of new inoculum for the following growing season.

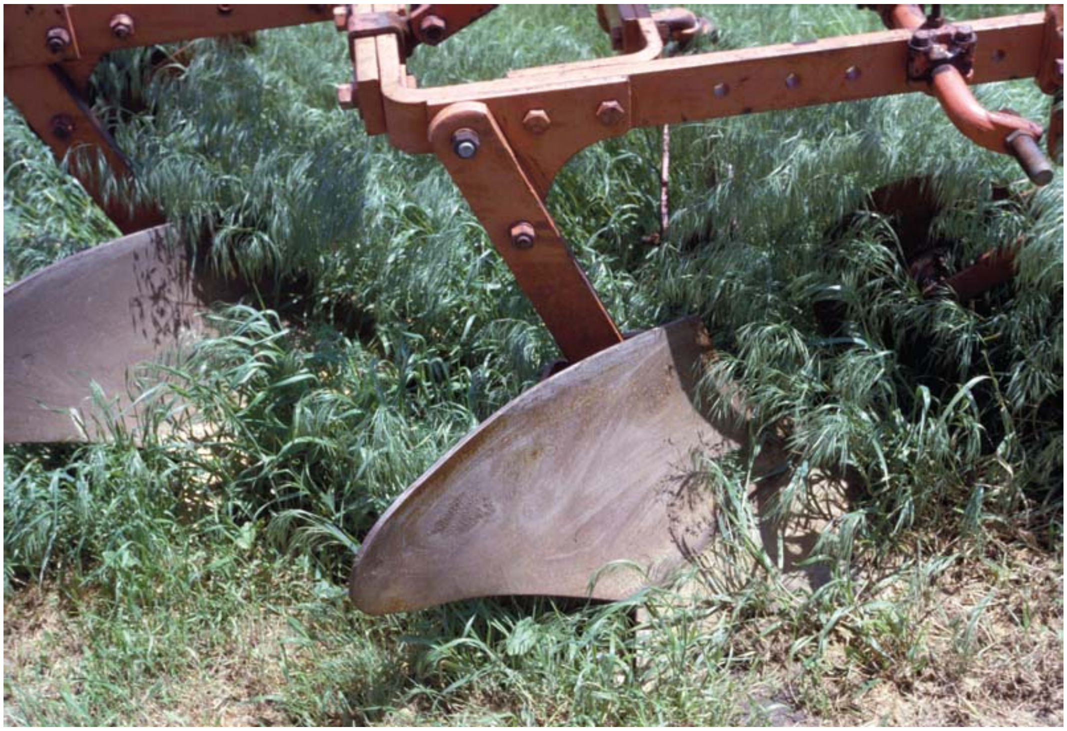


Figure 9.7. Tillage with a moldboard plow that turns stubble into the soil for degradation. The survival of pathogens is reduced when soil is tilled (plowed) to break up and bury infested plant debris.

Survival in Soil

Organisms that are well adapted to living as saprophytes in the soil are called **soil inhabitants**. As part of the normal soil microflora, their populations are fairly stable over time, although they can vary from year to year depending on environmental conditions. Most plant pathogens are considered **soil invaders** (or soil transients) when they live as soil saprophytes because they are not as competitive as the soil inhabitants. Their populations usually decline significantly over one or a few years in the absence of a host plant.

Soil inhabitants

- well adapted to living as saprophytes in the soil

Soil invaders (most plant pathogens)

- less competitive; populations drop relatively quickly in the absence of a host plant

The concept of the “soil invader” explains why crop rotation to a nonhost plant can be effective in reducing pathogen populations in the soil. In the absence of a host plant, the populations of biotrophs (obligate parasites) and soil invaders decline due to the absence of living host tissue and the presence of competition from soil microorganisms, respectively. In contrast, pathogen populations on perennial plants, such as orchard trees, strawberry, and turfgrasses, may continue to increase over time until the crops are no longer economically viable. The soil must then be planted with a different crop for several years, fumigated with chemicals, or treated with heat before it can be replanted with healthy plants of the previous crop species.

To help conserve soil and soil moisture, many growers have adopted no-till or minimum-till methods. This leaves a dense layer of stubble after harvest, which is an excellent way to reduce soil erosion and conserve soil water. Unfortunately, it also can help to protect pathogens from the competitive soil environment because the stubble is not broken up and buried. Fusarium head blight of wheat (caused by *Fusarium graminearum* and other species) (Figure 9.8A) and gray leaf spot of corn (caused by *Cercospora zeae-maydis*) (Figure 9.8B) are two diseases that became more severe following the adoption of reduced tillage practices.

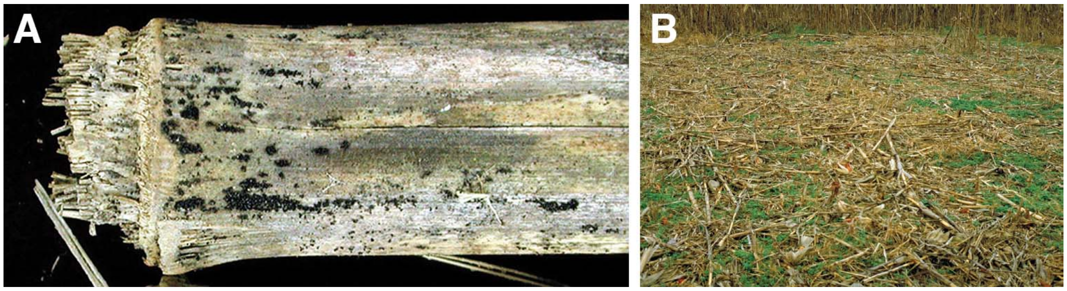


Figure 9.8. Pathogens surviving in plant debris. A, Perithecia of the Fusarium head blight pathogen, *Gibberella zeae* (anamorph: *Fusarium graminearum*), forming on the surface of infested plant debris. B, Corn debris infested by the gray leaf spot pathogen, *Cercospora zeae-maydis*.

Many plant pathogens survive well in plant debris, where there is less microbial competition than in soil. Removal or decay of infested debris helps to reduce pathogen populations. In soil, most pathogens are classified as soil invaders because they are less competitive than the resident microbial population in the soil (soil inhabitants). In the absence of a host, soil invader populations decline quite quickly. This is one reason that rotation to a nonhost crop effectively helps to reduce pathogen populations.

Survival Time of Plant Pathogens in Plant Debris or Soil

Days to weeks

- *Erwinia amylovora* (fire blight of pome fruits)
- *Tomato spotted wilt virus*
- *Xanthomonas axonopodis*, syn. *X. campestris* (citrus canker, bacterial spot of peppers)

Weeks to months

- *Gaeumannomyces graminis* (take-all root rot of turf and cereals)
- *Cronartium ribicola* aeciospores (white pine blister rust)

- *Hemileia vastatrix* urediniospores (coffee rust)
- eggs of *Meloidogyne* spp. (root-knot nematode)
- *Sclerotium rolfsii* (southern blight of many plants)

Months to a year or two

- eggs of *Heterodera glycines* (soybean cyst nematode)
- *Fusarium graminearum* (Fusarium head blight of wheat)

Many years

- *Fusarium oxysporum* (Fusarium wilt)
- *Phytophthora capsici* (Phytophthora blight of pepper)
- *Plasmodiophora brassicae* (clubroot of brassicas/crucifers)
- seeds of *Striga* spp. (witchweed)
- *Tobacco mosaic virus*
- *Verticillium* spp. (Verticillium wilt)

Survival as Epiphytes

Facultative plant pathogens, capable of living as either parasites or saprophytes, may grow and reproduce on living plant surfaces without infecting the host plant. Pathogens on plant surfaces are potentially vulnerable because they may be subject to desiccation and other environmental hazards, and they must compete with saprophytic organisms. Microorganisms that live on plant surfaces are called **epiphytes**. Epiphytes obtain nutrients from plant **exudates**.

EPIPHYTIC EXISTENCE ABOVE GROUND

An epiphytic existence is much more precarious on leaf surfaces than in the soil, because temperature and moisture fluctuations above ground are more variable. Because plants have evolved in a world filled with potential pathogens, some of their anatomical characteristics help to reduce the chance that pathogens will survive and penetrate. Foliar pathogens such as bacteria, fungi, and some nematodes require a film of water on the leaf surface for functions such as growth, movement, germination, and infection. The waxy surface of the cuticle helps water to run off the leaf, leaving a dry surface that is not conducive to an epiphytic existence.

Epiphytes: organisms that live on plant surfaces

Only limited aboveground plant structures are suitable **infection courts**. The guard cells of **stomata**, with their thinner and less waxy cuticle, as well as the stomata themselves, are vulnerable points on the leaf surface (Figure 9.9). However, in broad-leaved plants (dicots), most stomata are on the lower surfaces of leaves, where rainwater (or other moisture) is less likely to wet the surface compared to the top of a leaf. Some spores, such as rust urediniospores, infect leaves only through stomata (Figure 9.10). Bacteria and foliar nematodes also commonly infect through stomata.

Figure 9.9. Stoma (arrow) in a leaf surface, a common infection court for foliar plant pathogens (shown in a scanning electron micrograph).

Figure 9.10. Direct and indirect penetration of leaves by fungi (left) and bacteria (right).

Hydathodes are pores along the edges of some leaves where excess moisture can be exuded. When drier conditions resume, the exuded droplets, which may be contaminated by epiphytic bacteria or fungal spores from leaf surfaces, can be pulled into these more-vulnerable, thin-walled pores, where infection can occur. In moist conditions, the thin-walled parenchyma cells in **lenticels** on tubers and tree bark are vulnerable points for infection. **Wounds** provide both openings and nutrients for epiphytic pathogens. These wounds can be created by weather, human activities, and insect feeding and egg laying.

Aboveground Infection Sites on Plants

- **direct penetration**
  - epidermal cells or spaces between them
- **indirect penetration**
  - hydathodes
  - lenticels
  - stomata
  - wounds

The surface of the leaf that is influenced by plant exudates and the microclimate at the tissue surface and out to the **boundary layer** (see below) is called the **phylloplane**. Epiphytic organisms adapted to living on leaf surfaces are called **phylloplane-competent**. Some nonpathogenic fungi and bacteria are epiphytic residents of the phylloplane. These organisms compete with pathogens for food and space, produce antagonistic substances, and thus reduce foliar diseases. For example, powdery mildew fungi can germinate in high relative humidity, but they do not require free water as most fungal spores and bacteria do. Powdery mildews are more severe in humid weather without much rainfall, not only because the powdery mildew fungi can grow in these conditions, but also because their epiphytic fungal and bacterial competitors that require free water are less active.

Many pathogenic bacteria exist as epiphytes on plant surfaces and become pathogens only when environmental conditions are favorable for infection. In most field situations, it is highly unlikely that a single bacterial cell will successfully infect a plant, but bacteria on plant surfaces interact with each other through **quorum sensing** and a variety of signaling mechanisms. Quorum sensing allows a population of bacteria to behave more like a multicellular organism and increases the likelihood of successful infection. Thus, prevention of these diseases depends not on removing all of the epiphytic bacteria, which is too costly and difficult, but on predicting when infection is likely and trying to protect the plant during those events.

Many fungal, and especially bacterial pathogens, exist in low populations as epiphytes of aboveground plant surfaces and infect plants when conditions become conducive for disease. Hydathodes, lenticels, stomata, and wounds are important infection courts.

EPIPHYTIC EXISTENCE BELOW GROUND

Plant exudates are quite abundant in certain areas of the roots, especially where secondary roots break through cortical cells of the primary root and just behind the root cap, where meristematic cells actively divide and expand. The exudates from roots include amino acids, organic acids, and sugars. This nutrient-rich environment formed by exudates along root surfaces has been named the **rhizosphere** (Figure 9.11). The numbers and types of microorganisms found in the rhizosphere are different from those found in nonrhizosphere soil, i.e., soil that is usually more than 2 millimeters from the root surface and is not influenced by root exudates. About 12-40% (and sometimes much more) of the photosynthates produced by a plant are exuded into the rhizosphere. The plant benefits from this nutrient loss because the rhizosphere supports an active population of microorganisms that helps to protect the plant against invasion by pathogens.

Figure 9.11. A plant root and its rhizosphere (white area), the zone influenced by root exudates.

The importance of this protective microbial population was realized when plants were grown in completely sterile soil. Although the soil was sterilized to remove pathogens and protect the plants from infection, soil sterilization also killed the protective rhizosphere organisms. When a pathogen was accidentally introduced into the sterile environment, severe disease developed—more than would be expected in a nonsterile soil. This led to a modification of the soil sterilization process, called **soil pasteurization**, in which soil is heated to a temperature that kills most pathogens but not the protective soil inhabitants (see Chapter 11).

Organisms that are adapted to survival in the rhizosphere are called **rhizosphere-competent**. They are likely to be successful biological control organisms against root pathogens because they thrive in the rhizosphere and colonize the infection courts of root pathogens. Most root pathogens infect the natural wounds where lateral roots emerge or the area near the root cap where cells are rapidly expanding. These areas provide higher concentrations of root exudates to support both pathogens and protective rhizosphere organisms. The rhizosphere organisms compete with potential pathogens for both food and space. In addition, some species produce antibiotics and other antagonistic substances that inhibit competitors, including potential plant pathogens.

Root exudates create the nutrient-rich rhizosphere that supports a protective population of microbial soil inhabitants. Infection courts for pathogens, where lateral roots emerge or behind the root cap, also are the areas where root exudates are most concentrated. The protective rhizosphere organisms are potential biological control agents.

► **DID YOU KNOW?**

In the early 1900s, farmers believed that crop rotation was necessary to prevent the soil from becoming "worn out." When wheat was replanted into the same soil for several years, yields fell and plants grew poorly. Farmers blamed nutrient deficiencies, but in fact, these symptoms were mostly caused by soilborne pathogens. Diseased wheat roots and stubble helped pathogens survive, so disease increased, plant growth declined, and yields fell.

How do pathogens find plants?

Above Ground

The simple answer to this question is that pathogens usually have no active ability to seek out a plant. Fungi produce a variety of spores that are adapted for **passive dispersal through air** (Figure 9.12), but nearly all of the spores will fail to land on an appropriate host plant, which is why so many spores are required for survival. Once caught up in the moving air, spores can travel quickly and sometimes to surprisingly distant sites. Rust urediniospores have been trapped from airplanes in upper air levels. Soybean rust, caused by *Phakopsora pachyrhizi*, originated in Asia and spread to Africa by 1996. The disease was detected in South America in 2001 and was spread from there to the United States in 2004, probably by Hurricane Ivan.

Figure 9.12. Air dispersal of fungal spores. A, Forcible discharge of ascospores (resembling smoke) from the apothecia (fruiting bodies) of *Sclerotinia sclerotiorum* (white mold). B, Conidia of a powdery mildew fungus that have formed in chains to reach air currents. C, Conidia and conidiophore of *Botrytis cinerea* (gray mold). The conidiophore raises the conidia above the surface of the infected plant, plant debris, or soil to aid in air dispersal.

Fruiting structures on the surfaces of soil or on debris remaining from a previous growing season may **forcibly discharge** spores into the moving air masses. An example of this active form of dispersal includes the discharge of ascospores from asci in perithecia, ascostromata, and pseudothecia and on apothecia. Ascospores can be projected from 1 millimeter to up to 50 centimeters (20 inches), but most are discharged in the shorter end of the range. Basidiospores produced from the teliospores of rust and smut fungi and from the lining of the gills and pores of mushrooms and conks also are forcibly discharged from the cells that produce them. Basidiospores are generally projected only 0.05–0.1 millimeter. Spores must escape the calm layer of air (the boundary layer) on the surface of plants, plant debris, and soil in order to be carried away by more turbulent air.

Adaptations of Aboveground Pathogens

- air dispersal
- appressorium
- forcible discharge

- haustoria
- infection (penetration) peg
- phenological synchrony
- sticky spores or cells

Even though the probability of the successful infection by any single spore is very low, many pathogens improve their chances by producing large numbers of **asexual spores**, such as conidia and sporangia. Asexual spores are often produced soon after infection to help the pathogen exploit its food source by initiating new infection sites on the same plant or neighboring plants of the same species. Asexual spores on the infected plant tissue or infested plant debris may be produced in long chains or on conidiophores and sporangiophores that raise the spores above the surface, where moving air is more likely to carry them away. Later, the spores may settle by gravity, hit a surface, or be washed out of the air by rain.

Spores produced in a sticky substance (such as those that exude from acervuli or pycnidia) and sticky masses of bacteria rely on passing insects, water (primarily splashing rain or irrigation water), or human activities for travel to new plants (Figure 9.13). As with airborne spores, however, their movement is passive. Humans contribute to the spread of pathogens by pruning, cultivating, and performing other agricultural activities. Hands, machinery, and tools can carry sticky spores, bacteria, nematodes, and some viruses, which often are transmitted directly to another host plant.

Figure 9.13. Sticky fungal spores may be dispersed by splashing water, insects, machinery, or tools. A, Masses of sticky spores. B, Pycnidia exuding conidia in tendril-like masses of slimy spores (cirrhi) on the surface of an infected stem.

Insect or nematode **vectors** of pathogens are able to choose their routes and move to appropriate host plants; the pathogens are just along for the ride (Figure 9.14). Many vectors feed on a variety of host plants, so if the vector deposits a pathogen in or on a nonhost plant, the pathogen will be out of luck. The most common insect vectors of plant viruses are aphids, although other insects (including beetles, leafhoppers, mealy bugs, thrips, and whiteflies) can serve as virus vectors. The relationship between the virus and the insect vector varies. Some viruses have a short-term association with the vector (nonpersistent transmission) and others can remain in their vectors for several weeks or longer (persistent transmission) (see Chapter 5). In persistent transmission, the vector is likely to remain **viruliferous** for long periods and is able to infect more than one plant. This type of transmission is most important for long-distance dispersal of viruses.

Figure 9.14. When an aphid feeds on a leaf, it becomes a potential vector for a plant virus.

Some viruses are sap transmissible (also known as mechanically transmissible). Human activities, such as cultivation, and some natural events, such as hail and wind storms, can cause wounds and disperse plant pathogens in sap from one plant to the next. Fastidious bacteria (e.g., mollicutes and xylem-limited bacteria) are not sap transmissible, so their transmission is by insect vectors in nature and through plant propagation by humans.

Some pathogens that survive in the absence of their host have evolved the ability to track their host plants in time because they respond to the same environmental cues in their development. The term **phenological synchrony** is the coordinated development of pathogens and plants so that inoculum is produced when a plant is susceptible to infection. The rate of development is determined by the accumulation of heat units at the start of the growing season, known as **degree-days**. The rate of plant development and the rate of pathogen development are both governed by temperature;

development is faster on warm days and slower on cool days. The rate of development is sometimes modified by other environmental factors, such as moisture. In apple scab, the pseudothecia of *Venturia inaequalis* (Figure 9.15A) produce ascospores on fallen leaves from the previous season and discharge them when the new leaves of an apple tree begin to expand in the spring. The ascospores of the ergot fungus, *Claviceps purpurea*, are formed in ascostromata produced from ergots lying on the soil surface (Figure 9.15B) and are forcibly discharged when rye flowers are susceptible to infection. Basidiospores of the cedar-apple rust fungus *Gymnosporangium juniperi-virginianae* are produced on galls of a juniper tree when the leaves of the apple tree, the alternate host, are susceptible to infection.

Degree-Days

Degree-days **quantify** what we observe when we describe a year as having an “early” or a “late” spring. A base temperature is chosen that represents the beginning of development of a plant, insect, or other pest or pathogen in the spring. Each day, degree-days are calculated by determining the difference between the average temperature on that day and the base temperature. Degree-days accumulate more quickly in warm weather. No degree-days accumulate on days when the average temperature is below the base. Degree-day thresholds can be used to more precisely time monitoring activities and management practices.

Most pathogens are transported to new hosts by wind, water, or vectors. Asexual spore stages of fungi help the pathogens exploit food sources after successful infection by the survival spores or structures. Phenological synchrony describes the

coordinated development of pathogens and their host plants so that inoculum is produced when plants are susceptible to infection. Degree-days measure the heat units that affect both plant and pathogen development.

Figure 9.15. Phenological synchrony allows pathogens to produce inoculum at the time that host plants are susceptible to infection. A, A pseudothecium of the apple scab pathogen, *Venturia inaequalis*, from which ascospores are forcibly discharged during the period when apple leaves can be infected. B, A germinated sclerotium (ergot) (*Claviceps purpurea*) with ascostromata from which ascospores are forcibly discharged at the time that the host grass is flowering.

Below Ground

Soilborne pathogens are generally less mobile than above-ground pathogens. They may travel in dust blown by the wind, through the soil profile in moving water, or in soil attached to shoes, machinery, and harvested plant parts. Many soil-borne pathogens simply play a waiting game. A few are soil inhabitants and can survive as saprophytes until a host plant is nearby. Examples include *Pythium* spp., *Rhizoctonia* spp., and some soft rot bacteria. Even though soil invader populations may decline over time, they can rapidly increase if new host plants are successfully infected. In natural plant communities and with perennial hosts, soil invaders can usually rely on having a host plant in the following growing season.

Adaptations of Belowground Pathogens

- chemotaxis
- detection of host-specific root exudates
- fungistasis
- hyphopodia

- zoospore production

Nematodes and zoospores have some motility in soil. They are **chemotactic** and follow increasing concentrations of root exudates to vulnerable underground plant parts, especially the growing tips of roots (Figure 9.16).

Figure 9.16. Nematodes (arrow) in the root hair zone. Chemotaxis is the mechanism by which they find this zone.

Seeds of root-infecting parasitic flowering plants, eggs of nematodes, and spores of fungi may all find themselves in the soil under conditions of moisture and temperature that could trigger germination or hatching. If host roots are nearby, they should be successful in reestablishing their pathogenic relationship. Nematodes and zoospores have the advantage of motility and can move toward host roots, but a seed of a germinating parasitic flowering plant or a fungal spore may use up its stored food reserves before it can infect a plant if no host root is nearby.

► **DID YOU KNOW?**

In the dark confines of the soil, nematodes follow changes in temperature (thermotaxis) and chemical concentrations (chemotaxis) with their “noses” to find both food and mates.

Fungistasis is a phenomenon that keeps spores of pathogenic fungi from germinating too soon. Appropriate temperature and moisture alone do not stimulate spore germination; there is a nutrient requirement as well. The activities of soil saprophytes reduce the available nutrient levels in the soil below those needed to stimulate germination. This has been called an **exogenous dormancy** because active microbial populations in the soil impose the dormancy on the fungal survival structure. When the survival structure is in the proximity of an active rhizosphere (Figure 9.17), the high levels of nutrients in root exudates overcome fungistasis, so the survival structure can germinate. This nutrient signal is usually nonspecific, so nearly any plant's rhizosphere can stimulate germination. For other types of pathogens, a host-specific signal may be required. Some cyst nematode eggs and the seeds of witchweed (*Striga* spp.) require host root exudates from specific plants to stimulate hatching and germination, respectively. This prevents these biotrophs from becoming active in the absence of their host plants.

Figure 9.17. Pathogen activity near the rhizosphere. Note that fungistasis prevents germination of spores outside the rhizosphere. Nematodes and zoospores are attracted to the rhizosphere.

Rotation to a nonhost crop for a year or more helps to reduce the populations of soil invaders because they are forced to try to remain alive in a survival stage or in competition with saprophytes. In addition, the rhizosphere of a nonhost crop may overcome fungistasis and trigger pathogen activity. Crop rotation "tricks" potential pathogens into becoming active and using up their stored food reserves without providing a host plant that can support them. This is a very effective biological control method that can reduce populations of soil invaders.

How Rotation with a Nonhost Crop May Reduce Disease

- Soil invader populations decline in the absence of a host.
- Some pathogens require a host-specific root stimulus to germinate.

- Rhizosphere nutrients of nonhost plants can overcome fungistasis and “trick” fungi into germinating.

Nematodes and zoospores are motile, on a small scale, and use chemotaxis to find host plant roots. Fungi, bacteria, and parasitic flowering plants generally must wait passively for a host. The term *fungistasis* describes an exogenous dormancy imposed by soil saprophytes. The nonspecific nutrients of a plant rhizosphere can overcome fungistasis. This is one reason that rotation to nonhost plants can be successful in reducing fungal pathogen populations. Some pathogens require host-specific signals to resume active growth.

How do pathogens penetrate plants?

Once a spore or bacterium has successfully arrived on a leaf, it still faces the difficulties of invading that leaf. The waxy cuticle and cell wall components are barriers that keep almost all microorganisms from being plant pathogens. Some pathogens infect through wounds, where the initial physical barriers have been torn open and nutrients are more easily accessible. A few spores, such as rust urediniospores, can penetrate only through **stomata** (Figure 9.18). If placed on host parenchyma tissue from which the epidermal layer has been removed, the spore germ tube will search for a stoma until it starves to death without infecting the host despite being on host tissue. Scientists have tried to determine how germinating spores locate stomata on the leaf surface. They appear to orient their growth relative to the epidermal cell pattern and also to detect the height of stomatal guard cells. Chemical cues of gases emitted from stomata also play a role. If the epidermal pattern of the leaf could be changed, this might disorient rust spores and prevent infection.

Figure 9.18. The germ tube of a rust fungus spore grows to a stoma, produces an appressorium (arrow), and infects the leaf.

Many fungal pathogens, in particular, have evolved specialized structures to help them attach to and penetrate plant surfaces. Some fungi penetrate directly into epidermal cells or between them. These fungal pathogens often produce an **appressorium**, which is a specialized structure that helps to keep the spore from falling off the leaf (Figures 9.18 and 9.19). The appressorium "appresses" the germinating spore to the leaf surface with a sticky substance. A **penetration peg**, or **infection peg**, which is narrower than the spore germ tube, is sometimes formed and pushes into or between plant cells. Some fungi, such as *Rhizoctonia* spp., produce an **infection cushion** from which penetration pegs are produced. Below-ground, some soilborne fungi produce **hyphopodia** on roots; these are comparable to appressoria on leaves (Figure 9.20).

Figure 9.19. Diagrammatic view of spore germination. a = appressorium, g.t. = germ tube, i.p. = infection peg, h = haustorium.

Figure 9.20. Hyphopodia produced by a fungal pathogen (*Gaeumannomyces graminis* var. *graminis*) to infect roots. Note the infection pegs (arrows) in the centers of the lobes.

Until the mycelium is successfully inside the plant, it is vulnerable to desiccation and starvation. Once inside the plant tissue, it is less vulnerable to environmental extremes and has access to nutrients from the host plant without competition from saprophytes. Biotrophic fungi typically produce **haustoria** for nutrient absorption once they have overcome the surface barriers of the plant (Figure 9.19). Haustoria also are produced by parasitic flowering plants.

Nematodes and germinating seeds of parasitic flowering plants have enough pushing power to directly penetrate plant tissues.

Bacteria are not capable of penetrating plant tissue directly, so they seek points of vulnerability including natural openings (e.g., hydathodes, lenticels, stomata) and wounds. Fastidious bacteria and viruses rely mainly on vectors to penetrate their plant hosts.

Adaptations made by fungal plant pathogens to enable them to invade aboveground plant parts include appressoria, infection cushions, and penetration pegs for infection. Haustoria may be produced for nutrient absorption from host plants. Nematodes and parasitic flowering plants are relatively large and can apply mechanical pressure for infection. Parasitic plants rely on haustoria for nutrient absorption. Bacteria infect through wounds and natural openings. Fastidious bacteria and viruses usually require vectors to penetrate host tissues.

Part A. Ecological Interactions

Questions

1. What is a common survival strategy for each of these types of plant pathogens: fungi, bacteria, nematodes, parasitic flowering plants, and viruses?
2. What adaptations have foliar pathogens made to enable them to successfully infect leaves of a new host plant when they are in the soil or debris from the previous crop? How can we interfere with these processes to protect plants?
3. What is the rhizosphere and how does it affect plant health?
4. How do root pathogens find and infect plants? How can we interfere with these processes to protect plants?
5. Explain why tilling under old crop debris and rotation to a nonhost crop are successful methods of reducing pathogen populations. Describe a situation in which each method might not be practiced and what the effects might be.

Challenge Question

How does water affect plant disease? Consider its myriad effects on foliar pathogens, root pathogens, and host plants.

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

Ecological Interactions

alternate host
alternative host
appressorium (pl. appressoria)

boundary layer
chemotaxis
constitutive
cryptobiosis
degree-day
direct penetration
dormancy
epiphyte
exogenous
exudate
fungistasis
haustorium (pl. haustoria)
hydathode
hyphopodium (pl. hyphopodia)
indirect penetration
induced
infect
infection court
infection cushion
infection peg
infest
lenticel
melanin
penetration peg
phenological synchrony
phylloplane
phylloplane-competence
quorum sensing
rhizosphere
rhizosphere-competent
Sclerotium (pl. sclerotia)
soil inhabitant
soil invader

soil pasteurization
stoma (pl. stomates or stomata)
vector
viruliferous
wound

Part B. Physiological Interactions

IN THIS SECTION:

How do pathogens establish infections?

- **Enzymes**
- **Toxins**
- **Growth regulators (hormones)**
- **Polysaccharides**

How do plants defend themselves?

- **Constitutive (passive) defenses**
- **Induced (active) defenses**
- **Induced resistance**

How do pathogens establish infections?

Infection sites depend on whether the pathogen is invading above ground or below ground. Penetration may occur in two ways: **direct penetration** (i.e., through plant tissues) and **indirect penetration** (i.e., through natural openings or wounds). However, penetration does not necessarily lead to infection. Nonhost plants may be penetrated without further development of the pathogen.

Fungal, bacterial, and nematode parasites require nutrients from their hosts to grow and reproduce. In some cases, **latent** (or quiescent) **infections** occur, in which the pathogen does not fully establish itself until the correct environmental conditions or state of the host tissue occurs. For example, some fruit infections by fungal pathogens occur in the orchard but do not progress to a visible stage until after the fruits have been harvested and the tissue ripens.

During the infection process, there is a period of time between penetration and the first appearance of symptoms. Enough plant cells must be affected for the effects of the infection to become visible. This time is called the **incubation period**. It may be the same as or different from the **latent period**, which is the time required for the production of new inoculum. Some pathogens cause obvious necrotic symptoms, but it is often difficult to precisely determine the incubation period of infections by systemic pathogens such as viruses and fastidious bacteria. By the time symptoms appear, the pathogen is well established, and many preventive management practices will no longer be effective.

The primary goal of a plant pathogen is to reproduce itself without triggering plant defense reactions that are potentially fatal to it. Necrotrophs generally kill host cells and disable defense reactions. Biotrophs produce haustoria or otherwise obtain nutrients without triggering fatal defense reactions.

Pathogens may penetrate a plant directly or indirectly. Pathogens have successfully infected a plant when they obtain nutrients that allow them to grow and reproduce. There is usually an incubation period between infection and the development of visual symptoms.

Pathogens produce a wide variety of chemicals that contribute to their ability to successfully infect and colonize a plant and potentially cause disease. The most important chemical weapons of pathogens are enzymes, toxins, plant growth regulators (similar to animal hormones), and polysaccharides.

Enzymes

Enzymes are a requirement for life and are produced by all plants, animals, and microorganisms, including plant-pathogenic bacteria, fungi, nematodes, and viruses. Enzymes are proteins that function as catalysts for biological chemical reactions. In some cases, pathogen enzymes are similar or identical to those produced by saprophytes, which also must obtain food for growth and reproduction. Many of these enzymes are named for the substrate that they degrade, with the addition of the suffix *-ase*. For example, an enzyme that degrades cellulose is called *cellulase*, although there are different cellulases, which are produced by a variety of organisms.

Enzymes Produced by Plant Pathogens and the Compounds They Degrade

- cellulase (cellulose)
- hemicellulase (hemicellulose)
- pectinase (pectin)
- lignase (lignin)
- protease (protein)
- amylase (starch)
- lipase (lipid/fat)

Some enzymes degrade the host plant cell wall and other cellular components and are useful in digestion of complex molecules. The degradation of the plant cell wall provides nutrients for pathogens and also aids in the penetration and colonization of plant tissues. Bacteria and fungi secrete **extracellular** enzymes that externally digest plant cell components into smaller molecules that can be absorbed. In contrast, nematodes have an internal digestive system, although some plant-pathogenic nematodes (e.g., cyst and root-knot) secrete enzymes that degrade plant cell walls and aid in the colonization of host tissues.

Some enzymes are **constitutive** (produced at some level all the time). Others are **induced** (produced in response to some kind of signal or trigger), usually in the presence of a potential substrate. The production of enzymes requires metabolic energy, and living organisms do not want to waste energy producing enzymes when they are not needed.

Constitutive: always occurring or produced

Induced: occurring or produced in response to a stimulus

Constitutive enzymes can help an organism detect the presence of a food source. Many of the compounds found in plant cell walls are **polymers** (chains) of simple compounds that can be used as food for pathogens. For example, cellulose is composed of chains of glucose molecules. Pectin is a more complex molecule composed primarily of chains of galacturonan. Constitutive cellulases and pectinases (enzymes that degrade pectin) may produce **monomers** (single molecules) or **oligomers** (short chains) from these polymers that signal to the organism that the polymer is present. These smaller molecules induce the production of more and different enzymes by the pathogen. As is discussed later, these same molecules can be **elicitors** for defense reactions in the plant. This is one way that a plant can detect that it is being invaded.

Fungal and bacterial pathogens may produce **cutinases** (to degrade cutin in the cuticle), **cellulases, hemicellulases**, and **pectolytic enzymes**, or pectinases. Pectin is the primary component of the middle lamella that cements plant cells together. Pectinase producers often are soft rot pathogens that cause cell separation and tissue degradation; the plant tissues lose their integrity and become a slimy mess (Figure 9.21). Cell wall-degrading enzymes also are important in foliar spots and blights, root rots, and wilt diseases. Additional nutrients are obtained when organisms produce enzymes that degrade protein (proteases), lipids or fats (lipases), or starch (amylases).

Figure 9.21. Bacterial soft rot of melon, caused by *Erwinia carotovora* subsp. *carotovora* and the enzymes it produces.

Lignin is a phenolic component of secondary cell walls of plants and is the primary constituent that gives wood its strength. Because it is a complex molecule that is difficult to degrade, only a few specialized pathogens produce **lignase**, which allows them to become wood-decay organisms (Figure 9.22). Most of these organisms are basidiomycetes that produce relatively large fruiting bodies on the outside of their host trees and survive as saprophytes after the tree dies.

► **DID YOU KNOW?**

The chemical structure of lignin is similar to that of many waste products, such as PCBs, that persist in the environment. Lignin-degrading fungi have been used in industrial applications to degrade some of these compounds to reduce pollution.

Figure 9.22. Tree declining because of infection by a wood-decay fungus. Note the fruiting bodies (arrows) on the trunk of the tree. Wood-decay fungi produce lignases to degrade lignin.

Enzymes that catalyze the degradation of plant tissues aid in penetration, provide nutrients for the pathogen, and

contribute to symptom development. Enzymes that degrade cellulose, hemicellulose, cutin, and pectin are commonly produced. The smaller molecules produced by degradation of polymers such as cellulose and pectin can induce the pathogen to produce additional enzymes and can serve as elicitors of defense reactions by the plant. Only some specialized pathogens produce enzymes that degrade lignin.

Toxins

A number of plant pathogens produce **phytotoxins** (toxins that are poisonous to plants). These toxins are clearly special adaptations for pathogenic organisms because toxins poison host plant cells and are unnecessary for a saprophytic existence. Toxins are typically produced by necrotrophs because biotrophs require living plant cells. Toxins contribute to the development of chlorosis and necrosis in infected plant tissues. They can be divided into two broad groups: general and host specific. Their roles are quite different.

General toxins: virulence factors

- contribute to disease severity but are not necessary for disease to occur

Host-specific toxins: pathogenicity factors

- required for infection

General or **nonspecific toxins**, as their name suggests, are toxic to most plants. They seem to aid the pathogen, but mutants that are unable to produce the toxin are still pathogenic. Thus, general toxins appear to be **virulence factors** that contribute to the severity of the disease that occurs. When host cells are poisoned, biological defense reactions can be inhibited or halted. Toxins produced by bacteria (e.g., phaseolotoxin, tabtoxin) and fungal species in the genus *Fusarium* are general toxins.

Host-specific toxins are more specialized and are toxic only to plants susceptible to the pathogen that produces them. If a pathogen loses its ability to be pathogenic when a genetic mutation removes its capacity to produce a host-specific toxin, the toxin is a **pathogenicity factor**, essential for disease to occur. If a pathogen can still cause disease, although at lesser severity, even when it does not produce a particular host-specific toxin, the toxin is termed a **virulence factor**.

Host-specific toxins are less common than general toxins. They may pose a new threat when new genetic lines of host plants become popular. For example, in the 1940s, an oat cultivar named Victoria was used as a parent for many new oat cultivars, including some in the United States, because it had excellent resistance to crown rust and smut. Surprisingly, Victoria oats were highly susceptible to a newly discovered fungus that was given the name *Helminthosporium victoriae* (renamed *Bipolaris victoriae*; teleomorph: *Cochliobolus victoriae*). This led to the first discovery of a host-specific toxin, which was named victorin (or HV toxin). Leaf blight symptoms could be induced in Victoria oats using purified preparations of victorin, even in the absence of the fungal pathogen.

Phytotoxins, produced by necrotrophs, poison plant cells. General toxins affect a wide variety of plants and contribute to the ability of a pathogen to infect a plant and cause disease. Host-specific toxins are pathogenicity factors required for successful infection by the pathogen.

Another example of a similar problem occurred in 1970. The hybrid seed corn industry had discovered a genetic factor that gave male sterility to corn plants. This was a great boon to the industry because it meant that corn no longer had to be detasseled by hand, which saved labor expenses. Detasseling removes the pollen-producing (male) part of corn and prevents accidental self-pollination of the plants that serve as the female parent in the production of high-yielding hybrid seed. The genetic factor was known as Texas male-sterile cytoplasm (TMS) and was associated with genes in the mitochondria.

► **DID YOU KNOW?**

It is likely that two hurricanes played a significant role in the spread of the southern corn leaf blight pathogen in 1970. The disease was first reported in Florida in late April and early May. Hurricane Alma moved across Florida to the Gulf of Mexico coast from May 17 to 27. The next disease reports were in the Gulf Coast states in early June. That same year, Hurricane Becky moved from the Gulf of Mexico up the Mississippi River valley from July 19 to 23, the same pattern in which the disease then developed. (Information courtesy W. Pedersen, University of Illinois)

Following the widespread planting of TMS corn, a new race of the southern corn leaf blight pathogen, *Bipolaris maydis* (teleomorph: *Cochliobolus heterostrophus*), was discovered. The new race T was much more aggressive than the original race O because it produced T-toxin, a host-specific toxin (Figures 9.23 and 9.24). Although race T could infect corn with normal cytoplasm, T-toxin affected only TMS corn, resulting in severe disease. Losses of nearly $2.5 billion were caused by this new pathogen race. Race T grew best at higher temperatures, and losses in southern states were 100% on some farms.

Figure 9.23. Corn infected with race T of *Bipolaris maydis*, which produces a host-specific toxin. In the southern United States, the 1970 southern corn leaf blight epidemic destroyed the corn crop on many farms.

Figure 9.24. A corn plant with Texas male-sterile cytoplasm (left) is very susceptible to the host-specific toxin produced by race T of the southern corn leaf blight (SCLB) pathogen. Corn with normal cytoplasm (right) is much less susceptible to SCLB.

Later research determined that the T-toxin attached to a physical site on the mitochondria of TMS corn that did not exist in corn with normal cytoplasm. Genes associated with mitochondria or chloroplasts are called **extrachromosomal** or **maternal genes**. These genes are passed through the cytoplasm of maternal lines during sexual reproduction, a process called **cytoplasmic inheritance**. This was the first example of the impact of cytoplasmic inheritance on susceptibility to a plant disease.

Cytoplasmic inheritance = Maternal inheritance = Extrachromosomal inheritance: transmission of genes located in mitochondria and chloroplasts (not the nucleus) during mating

► DID YOU KNOW?

Inheritance associated with genes of organelles is sometimes called “maternal” inheritance because males contribute primarily nuclear genes in mating. The female sex cell includes both a nucleus and organelles in the cytoplasm. The genes in your mitochondria can be traced back to your mother and her mother and so on.

Growth Regulators (Hormones)

Growth regulators are molecules that impact growth processes in plant tissues and are active at very low concentrations. Even small changes in the levels of specific growth regulators or their balance in the plant can cause significant changes in growth patterns. Pathogens affect the levels of growth regulators in plants by producing these compounds directly or by causing the plant to produce more or less growth regulator than normal. This ability, like that of toxin production, is an adaptation for parasitism.

Symptoms associated with growth regulator changes include defoliation, etiolation, galls, leaf roll or curl or other deformations, stunting, and witches' brooms. Disease names sometimes reflect these symptoms: black knot, clubroot, crown gall, potato leafroll, or root knot. Note that these diseases are caused by diverse organisms: a fungus, a plasmodiophoromycete, a bacterium, a virus, and a nematode, respectively. As in animals, it is the interaction of growth regulators that results in normal or abnormal plant growth and development.

Many gall symptoms are caused by an overproduction of **auxins** and **cytokinins**. When a gall forms, plants produce both more cells **(hyperplasia)** and larger cells **(hypertrophy)** to create a site for spore production, nutrition, or shelter for the pathogen. The crown gall bacterium inserts auxin- and cytokinin-producing genes from its Ti (tumor-inducing) plasmid into the plant genome—an example of natural cross-kingdom genetic engineering. Root-knot nematodes (Figure 9.25A) and the pathogens that cause cedar-apple rust, clubroot, and corn smut also induce plants to form galls, although not by gene transfer.

Plant Growth Regulators Produced by or Affected (Increased or Decreased) by Pathogens

- auxins
- cytokinins
- ethylene
- gibberellins

Figure 9.25. Symptoms of plant diseases involving disruption of normal levels of growth regulators. A, Root galls and growth distortions of carrot caused by root-knot nematodes (*Meloidogyne* spp.). B, Rice with foolish seedling (bakanae) disease. Note etiolation of infected seedlings on the right due to gibberellin production by *Gibberella fujikuroi*. C, Summer squash infected by *Cucumber mosaic virus*. Note stunting and severe leaf distortion. D, Rose leaf with yellowing from ethylene production by a fungal pathogen, *Diplocarpon rosae* (black spot disease). E, Witches' broom of alfalfa caused by a phytoplasma. Note the proliferation of stems and the pale foliage.

The foolish seedling disease of rice (also called bakanae from a Japanese word) is caused by the fungus *Gibberella fujikuroi* (anamorph: *Fusarium moniliforme*), which causes etiolation, or elongated growth of the stems. Infected seedlings grow so tall that they fall over (Figure 9.25B). Studies of this disease led to the discovery that plants produce growth regulators that are chemically similar to those produced by this fungus, so they were named **gibberellins**. Stunting, a common symptom of infection by viruses and fastidious bacteria, can be overcome by application of gibberellins to plants, suggesting that these pathogens may inhibit normal gibberellin production (Figure 9.25C), since viruses do not produce any growth regulators directly.

Some fungi and bacteria produce **ethylene**, a growth regulator that affects leaf abscission, fruit ripening, and plant senescence, among other functions. For example, the fungus that causes black spot of rose (*Diplocarpon rosae*) produces ethylene that induces chlorosis of the leaves and eventually leaf **abscission** (or drop) (Figure 9.25D). Ethylene production by pathogens also plays a role in vascular wilt diseases.

► **DID YOU KNOW?**

Stomata have traditionally been considered vulnerable areas for invasion by bacteria. Recent research suggests that plants actually close their stomata as a defense against bacterial invasion. In response, some bacteria produce chemicals similar to the plant growth regulator jasmonate that can cause the stomata to reopen.

Dwarf mistletoes, phytoplasmas, powdery mildew fungi, and other pathogens can disrupt the normal branching patterns of plants, especially trees, leading to the formation of witches' brooms (Figure 9.25E). A proliferation of roots is a similar symptom that is caused by several kinds of pathogens. A number of viruses disrupt normal leaf growth patterns, resulting in leaf curl, leaf roll, or rugosity.

Pathogens produce plant growth regulators directly or affect their production by the plant, either increasing or decreasing normal amounts. Auxins, cytokinins, gibberellins, and ethylene are commonly affected. Symptoms due to growth regulator imbalances include etiolation, galls, leaf distortions, stunting, and witches' brooms. Modification of plant cells and plant growth patterns can enhance nutrient absorption or provide a living site for the pathogen.

Polysaccharides

Fungi and bacteria often appear slimy due to the production of extracellular polysaccharides. These substances probably aid in pathogen survival by helping to prevent desiccation. Polysaccharides also are believed to play a role in pathogenesis for some pathogens, especially bacteria. Polysaccharide production contributes to the mechanical blockage of xylem tissue in vascular wilt diseases.

Polysaccharides have a role in pathogenesis for some pathogens. They can contribute to mechanical blockage in vascular wilts.

How do plants defend themselves?

Constitutive (Passive) Defenses

Constitutive (Passive) Plant Defenses

Structural

- bark
- bud scales
- collenchyma
- dense trichomes (hairs)
- sclerenchyma
- stomata on lower leaf surface
- suberized outer layers of bulbs, tubers, etc.
- waxy cuticle

Chemical

- cyanogenic glucosides
- phenolic glucosides
- proinhibitins
- secondary metabolites

Pathogens have evolved a variety of weapons to aid their invasion of plants. Although it may not be obvious, plants respond to invasion with a variety of defenses. Plant defenses may be structural or chemical. Some defenses are constitutive (always present) and others are induced.

Some of the **constitutive structural defenses** are familiar features of plants: a waxy cuticle (Figure 9.26); the size and shape of stomata on lower leaf surfaces, bark, and bud scales; dense layers of hairs (trichomes); suberized cells in the outer layer of tubers (Figure 9.27), corms, and bulbs; and lignification of woody tissues. Vascular bundles are protected by tissues with thickened walls, such as **collenchyma** and **sclerenchyma**. The outer cell walls of primary roots usually include cutin and suberin, but this is not as obvious as the cuticle seen on leaves. In contrast, the root hairs and apical meristems of roots are quite vulnerable tissues. Older roots of woody plants develop bark. These many protective anatomical structures help exclude saprophytes, but plant pathogens have evolved to infect plants despite them.

Figure 9.26. The waxy surface of these bell peppers helps reduce surface water and infection by fungi and bacteria.

Figure 9.27. Most surface cells of potato tubers are suberized and provide a good defense against many pathogens. Potato tuber lenticels (arrow) are usually inconspicuous, but they become enlarged in wet soil, allowing entry by pathogens.

There has been much speculation about the constitutive chemical content of plant cells and its impact on invading pathogens. There are clearly some differences in nutrient content, pH, and toxic compounds in the cells and tissues of different plant species, but all plants are susceptible to pathogens, so these contents are not obviously linked to resistance in most cases.

At the biochemical level, many plants produce **secondary metabolites** that are not necessary for the growth of the plant; thus, they must have other functions (Figure 9.28). Caffeine, cocaine, and nicotine are well-known examples of secondary metabolites in plants. Some of these chemicals probably aid in resistance to insect pests, and some play a role in defense against pathogens. Some of these **constitutive chemicals** are known as **proinhibitins** because they help to defend a plant if a microorganism invades it. For example, some plants produce **glucosides**, which are toxic compounds that are bound to sugar molecules. The toxic components may be cyanogenic (releasing cyanide) or phenolic. Upon invasion by a pathogen, the toxic portion of the molecule is released, poisoning the invader. Although these are likely to help prevent the invasion of some potential pathogens, several pathogens of these plants are successful because they have developed a means to detoxify the poisons.

Figure 9.28. Onion smudge, a fungal disease, on a white onion. Red onions are more resistant to smudge because of the chemical inhibitors in the colored bulb scales.

► DID YOU KNOW?

Naturally toxic compounds in plant tissues were discovered in a classic study of onion smudge disease caused by the fungus *Colletotrichum circinans*. When the bulb scales of colored onions are wet, toxic compounds diffuse out of the scales and inhibit germination of the fungal spores. White onions, which lack these compounds, are susceptible to smudge. (See Figure 9.28.)

Plants have a variety of constitutive (passive) structural and biochemical defenses. These contribute to the inability of most microorganisms to infect plants. Plant pathogens have evolved ways to infect their host plants despite these defenses.

Induced (Active) Defenses

Induced (Active) Plant Defenses

Structural

- abscission layers
- cork layers
- gums or resins
- papillae of callose
- thickening and/or lignification of cell walls
- tyloses

Chemical

- pathogenesis-related (PR) proteins
- phytoalexins

As described previously, the very early events in the interactions between plant hosts and pathogens often involve cell wall degradation by pathogen enzymes. The resulting monomers and oligomers of the polymer constituents can serve as triggers or inducers for new and different pathogen enzymes. These same molecules may be **elicitors** of **induced defenses** in the plant.

The early stages of plant defense activity can be detected at the cellular level. If adenosine triphosphate (ATP) production is monitored, a sharp spike in ATP production is seen (as part of a general increase in respiration) soon after infection and even before symptoms can be observed. This ATP spike is primarily due to the need for energy to induce the chemical and structural defenses that make up the plant's response to infection. Microarray data and transcriptional profiles from recent research suggest that a cellular reprogramming goes on in response to infection. The plant cells must shift from processes associated with growth and photosynthesis to others associated with secondary metabolism and defense. Transcription and translation activities increase as genes become active for the production of enzymes and other induced defense compounds.

A number of **structural defenses** are induced by various kinds of plants when they are invaded. Cell wall changes are commonly observed during invasion by fungi or bacteria. Cell walls thicken and sometimes lignify, which helps to prevent multiplication of bacteria and further invasion by fungi. **Papillae** composed of **callose** form on the inside of the cell wall and surround hyphal tips that are attempting to penetrate (Figure 9.29A). Papillae may become lignified, which makes the papilla walls very resistant to the cell wall-degrading enzymes produced by pathogens. This appears to stop or delay invasion.

Figure 9.29. Some examples of induced structural defenses. A, Papilla (arrow) formed around a fungal hypha at the site of attempted infection. B, Longitudinal section of an elm vessel with tyloses (arrow) that formed in response to Dutch elm disease. C, Resin (arrow) production in conifer with Armillaria root disease. D, Shot-hole symptom in tobacco, caused by

the fungus *Cercospora nicotianae*. The formation of a circular abscission layer causes the infected part to fall out.

Tyloses are parenchyma cells that balloon into xylem vessels through the pits in the xylem secondary wall (Figure 9.29B). These commonly form in response to vascular wilt diseases and contribute to disease symptoms by blocking the xylem. They also serve as a defense mechanism when a woody branch is cut or injured, to help reduce the loss of xylem fluid or to defend against invasion by pathogens. The rapid development of tyloses following root infections by vascular wilt pathogens may block the pathogen effectively enough to serve as a resistance mechanism. **Gums** in peach and cherry trees and **resins** in conifers create wound barriers against invading pathogens (Figure 9.29C).

Entire tissues can be modified or induced as a defense. Cork cells form after wounding or invasion of tubers, other storage organs, and even leaves. **Cork layers** and **abscission layers** of lignified cells help the leaves to block pathogens from further invasion. In the case of the **shot-hole** symptom, the infection site is eliminated from the plant when a circular abscission layer causes the infected piece of leaf tissue to actually fall out (Figure 9.29D).

Biochemical defenses also are produced by plants under attack. Plants may detoxify toxic compounds or inhibit enzyme activity of invading pathogens. Plants produce enzymes that inhibit invaders. For example, plants may produce chitinases that degrade chitin in fungal cell walls or glucanases to degrade β-glucans in oomycete cell walls. In experiments, the addition of genes to plants to increase their ability to produce such enzymes also increases their disease resistance. **Pathogenesis-related proteins (PR proteins)** are the various enzymes and other proteins produced as defense compounds by plants. The function of many of these proteins is not yet known.

Examples of Pathogenesis-Related (PR) Proteins

- chitinases
- gluconases
- proteases

Phytoalexins are an important group of induced antimicrobial plant compounds. They are defense compounds that are toxic specifically to the pathogens of the host plant. They are often named for the Latin name of the host, for example, capsidiol in peppers, phaseolin in beans, and pisatin in peas. Phytoalexins may be triggered by cellular invasion due to pathogens, insect pests, or wounds.

Phytoalexins

- induced antimicrobial compounds
- induced by herbivores, insects, microorganisms, wounding, and certain chemicals
- plant specific, but active against a wide range of organisms

There is evidence that phytoalexins are important defense compounds. Pathogens that infect specific plants often are able to degrade or inactivate the phytoalexins of their host. In an interesting experiment, a gene for a pisatin-inactivating enzyme of a fungal pea pathogen was added to a corn pathogen, and the corn pathogen became capable of infecting peas. The added gene allowed the pathogen to expand its host range. When the same gene was added to a fungal saprophyte, however, it did not become a plant pathogen. Although the genes that confer these individual defenses play a role in host-pathogen interactions, the ability to be a pathogen is a complex adaptation that involves more than a single gene.

► DID YOU KNOW?

The word *phytoalexins* was created from Greek words that mean “plant protectors” to describe the protective function of these chemicals.

Researchers have been very interested in the earliest moments of the interaction between hosts and pathogens. Molecular signals or elicitors on pathogens are recognized by plant receptors. The term **pathogen-associated molecular patterns**, or **PAMPS**, is sometimes used to describe these small molecular motifs. Because most microbes have these motifs, a more general term, **microbe-associated molecular patterns**, or **MAMPS**, is sometimes used. In addition to PAMPS (and MAMPS), there also are endogenous (plant-derived) elicitors that are produced when pathogens interact with host cell walls, triggering plant defenses.

All plants have defenses. The survival of the plant often is dependent on how quickly and how strongly it can deploy these defenses. Much of the defense reaction is a biological response, so stress factors can limit a plant’s ability to defend itself. This is one reason that stressed plants tend to be more susceptible to disease. Important abiotic stress factors that can delay or reduce a plant’s defense reactions include air pollution, drought, heat, nutrient deficiencies, salinity, soil compaction, and water-logged soils with little oxygen.

► DID YOU KNOW?

Willow bark has been used in folk medicine for centuries as an analgesic and fever reducer. Salicin is the active ingredient in willows (*Salix*) from which acetylsalicylic acid (aspirin) was derived. The methyl ester of salicylic acid is methylsalicylate, sometimes called oil of wintergreen, which is volatile and may serve as a signal between plants.

Induced defenses are produced before symptoms can be observed. Structural defenses may develop at the cellular or tissue level. Biochemical defenses include an array of pathogenesis-related (PR) proteins, especially enzymes, and phytoalexins.

Induced Resistance

Because many of the important structural and biochemical defenses of a plant are induced, plant pathologists have wondered if it would be possible to preemptively induce these defenses as a way to protect plants from infection. In the 1930s, K. S. Chester summarized the evidence that prior infection of plants could lead to an increase in resistance to other pathogens at or at some distance from the initial infection sites. This phenomenon was termed **induced resistance.** This type of resistance was clearly demonstrated in the 1960s and 1970s using tobacco and *Tobacco mosaic virus* (TMV) and the anthracnose fungus, *Colletotrichum lagenarium*, on cucumbers. When one leaf of tobacco was infected with TMV or a leaf of cucumber was inoculated with the anthracnose fungus, localized necrotic lesions formed on the inoculated leaves. In response to these initial inoculations, the plants generated a translocated signal that induced resistance throughout the plants. Like animals activating their immune systems, plants can turn on their defenses and protect themselves against further infection. Very different from the specificity of animal immunity, however, this defense reaction is general and effective against a variety of pathogens and pests, including bacteria, fungi, insects, nematodes, and viruses.

How can invasion of a plant in one location signal defense reactions in other parts of the plant? Studies have shown that the signal that is transported throughout plants is associated with salicylic acid. Additional studies have shown that, although salicylic acid plays a role in triggering the signal, it is not the actual signal. Additional compounds involved in defense signaling include jasmonic acid, ethylene, and peroxidases. The development of resistance following a challenge inoculation by a pathogen was named **systemic acquired resistance** (**SAR**). This type of resistance elicits the death of one or a few cells, known as the **hypersensitive response (HR)**, and production of PR proteins, and it can be triggered by bacteria, fungi, nematodes, and viruses (Figure 9.30). A similar form of acquired resistance that does not require the HR or even contact with a pathogen can be induced by the rhizosphere bacteria that promote plant growth. This has been called **induced systemic resistance** (**ISR**). It apparently induces certain structural defenses and phytoalexins, but not the PR proteins of SAR.

Systemic Acquired Resistance (SAR)

- Induced by a pathogen triggering the hypersensitive response (HR) and the production of certain chemicals such as pathogenesis-related (PR) proteins
- Signal is sent to distant tissues to turn on defenses (may involve salicylic acid, jasmonic acid, peroxidases, ethylene).
- Distant tissues are protected from a variety of pathogens and even insects.

Figure 9.30. Systemic acquired resistance (SAR) is induced in the new growth of a plant that has been exposed to a pathogen.

Induced Systemic Resistance (ISR)

- similar to systemic acquired resistance (SAR) but induced by nonpathogens such as rhizobacteria
- antimicrobial pathogenesis-related (PR) proteins not produced

One might wonder why these defenses are not always active if they are so effective. As with many enzymes and other induced compounds, the continuous production of unnecessary defenses would be a waste of the plant's energy.

It is not practical to give plants "a little disease," so researchers have discovered chemicals that can be used to induce SAR. These chemicals are often called "plant activators." One commercial product, Elexa, is said to bind to fungal receptor sites on the plant and to elicit plant defense responses. Another commercial product, Actigard (acibenzolar-*S*-methyl), belongs to a new chemical class known as the benzothiadiazoles that are systemically transported throughout the plant and trigger SAR.

Plant activators: commercial products that are applied to plants to trigger systemic acquired resistance (SAR)

Induced resistance occurs when infection triggers defense reactions in cells and tissues that have not yet been infected. It is related to the plant's ability to detect invasion and to

cause the death of one or a few cells at the point of infection, known as the hypersensitive response (HR). Systemic acquired resistance (SAR) occurs in distant tissues and new growth following HR or contact with a pathogen; it can be triggered by a variety of pathogens and certain chemicals. Induced systemic resistance (ISR) is a similar response that can be triggered by rhizosphere bacteria. These forms of resistance are effective against a variety of pathogens.

Although commercial products are already in use, much remains to be learned about how stable, reliable, and persistent the defense reactions that they induce will be. It also is important to understand when and how often these products need to be applied to plants and what effects they may have on the yield and quality of plant products. The ability of these new products to improve yields and to protect plants from disease requires more study, but it is a very exciting approach to plant protection.

A Chess Game Analogy

A chess game analogy has been used to describe the interactions between plants and their pathogens. In the same way that each player counters the other’s moves in a chess game, the plant and pathogen defend themselves against the other’s attacks. A pathogen produces a toxin, and the plant discovers a way to detoxify it. The plant produces a phytoalexin, but the pathogen becomes able to inactivate it. These interactions are part of the evolutionary relationships between hosts and pathogens over time.

Part B. Physiological Interactions

Questions

1. Name the three major types of chemical weapons used by pathogens of plants. What symptoms would you expect for each?
2. What are three common enzymes produced by plant pathogens for degradation of cell walls? Why are some enzymes produced constitutively but others are induced?
3. What is the difference in *function* between a general toxin and a host-specific toxin in pathogenesis?
4. What are some common symptoms of diseases caused by biotic pathogens that cause changes in the amount or the balance of growth regulators in plants?
5. Name several structural defenses and chemical defense compounds produced by plants. Do susceptible plants lack the ability to produce defense compounds? Explain.
6. The interaction between hosts and pathogens has been described as a "chess game." Explain this analogy. How can cultural practices and environmental conditions modify the outcome of such an interaction?
7. Systemic acquired resistance (SAR) shows promise for disease management. Explain what SAR is and how it can be induced. What factors can modify SAR? Is SAR likely to replace the use of fungicides for diseases caused by fungal pathogens?

Challenge Question

Describe the basic *biology* (type of organism, reproduction, nutrition) of the major groups of plant pathogens and what *adaptations* they have made to become pathogens of plants.

Consider, for example, survival, dispersal, penetration, and infection.

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

Physiological Interactions

abscission
auxin
callose
collenchyma
constitutive
cork
cytokinin
cytoplasmic inheritance
direct penetration
elicitor
enzyme
ethylene
extracellular
extrachromosomal inheritance
gibberellin
glucoside
growth regulator
gum
hyperplasia
hypersensitive response (HR)
hypertrophy
hyphopodium (pl. hyphophodia)
incubation period
indirect penetration

induced
induced systemic resistance (ISR)
latent infection
latent period
lenticel
maternal inheritance
microbe-associated molecular patterns (MAMPS)
papilla (pl. papillae)
pathogen-associated molecular patterns (PAMPS)
pathogenesis-related (PR) proteins
pathogenicity
phytoalexin
proinhibitin
resin
sclerenchyma
secondary metabolite
shot-hole
systemic acquired resistance (SAR)
toxin
tylosis (pl. tyloses)
virulence

Part C. Genetic Interactions

IN THIS SECTION:

What are the genetic interactions between plants and their pathogens?

- **Gene-for-gene interactions**

How can we create disease-resistant plants?

- **Genetic variation in plants and their pathogens**
- **Specific (R gene) resistance**
- **General (race-nonspecific) resistance**
- **Biotechnology and genetic engineering for disease resistance**
- **Optimizing resistance to pathogens**

What are the genetic interactions between plants and their pathogens?

Disease is actually a relatively rare event that requires a compatible genetic interaction between a host plant and a pathogen. For example, potato plants do not get Dutch elm disease under any circumstances. Specific potato cultivars may be susceptible only to specific races of the late blight pathogen, *Phytophthora infestans*. As described previously, the pathogen usually produces **elicitors** that are recognized by the **receptors** in the host that trigger resistance responses. We use the words **resistant** and **susceptible** to describe the range of reactions in the plant when hosts and pathogens interact. Plants may be highly resistant (Figure 9.31) or only moderately so. If a plant does not become diseased simply because of the vagaries of weather, vector movement, or other unexplained causes, its lack of disease is considered an **escape** (Figure 9.32).

Sometimes plants develop disease with little or no impact on the final yield, an interaction called **tolerance**.

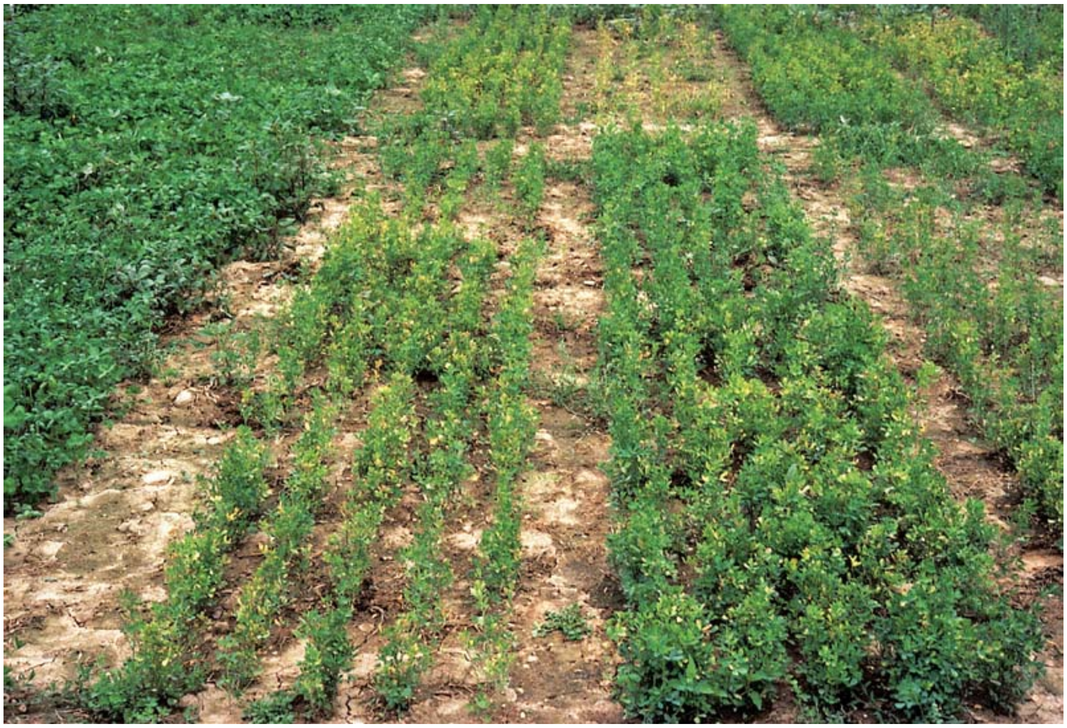


Figure 9.31. Alfalfa lines susceptible (left) and resistant (right) to Aphanomyces root rot in naturally infested soil.

Figure 9.32. An elm tree that has escaped Dutch elm disease despite the death and removal of many nearby elms.

Host Reactions to Pathogens

- resistance
- susceptibility
- escape
- tolerance

People have been trying to select disease-resistant plants since agriculture began, long before anything was known about the genetics of host-pathogen interactions. By saving seed of plants that had survived epidemics, farmers hoped to obtain plants that might survive future epidemics.

Some of our worst epidemics have occurred when an exotic pathogen was introduced into a plant population that had little or no natural genetic resistance. Citrus canker, chestnut blight, Dutch elm disease, dogwood anthracnose, pine wilt, Ramorum blight (sudden oak death), and soybean rust are important examples in the United States. In some cases, the introduction of a new genetic factor, thought to be unrelated to disease, can lead to an epidemic. The 1970 southern corn leaf blight epidemic of TMS corn (described in Part B) was the most disastrous single-year epidemic in U.S. history. For most endemic diseases, some losses occur each year, and environmental conditions play an important role in disease severity.

Pathogenicity is the ability of a pathogen to cause disease of a plant species. An organism is either capable of infecting that host (in which case, it is a pathogen) or it is not. Just as plants can vary in their resistance, pathogens can vary in their ability to cause disease. There are biotypes, races, or strains of pathogens that vary in their ability to infect different cultivars of the same plant species because of the resistance genes these cultivars have. This may be a qualitative reaction (disease or no disease, +/–) or a quantitative one ranging from minor disease to severe disease. These variable reactions are described by the word **virulence**. Confusingly, *virulence* has been used to describe three different characteristics of host-pathogen interactions: host damage; pathogenicity of a particular biotype, race, or strain of an organism; and gene-for-gene interactions, as explained in the following paragraphs.

One common use of the term *virulence* is to **quantify** pathogenicity in terms of host damage. Different isolates may be more virulent than others under the same conditions. More virulent strains often have a shorter latent period and/or produce new inoculum more quickly and/or in greater amounts.

Virulence is sometimes used to describe which pathogen races can infect which plant cultivars. It is used as a **qualitative** description of their ability to cause disease. For example, *Phytophthora infestans* is pathogenic on potatoes. Race 1 of *P. infestans* is virulent on potato cultivars with the R1 gene, but race 0 is not. The word *pathogenic* could be used instead of *virulent* in this situation.

The most confusing use of the term *virulence* lies in its use with the term *avirulence* in gene-for-gene interactions. The word *avirulence* seems to suggest that the pathogen is not virulent, but it really indicates the compatibility or incompatibility of the interaction between a host cultivar and a pathogen race (see below).

In practical terms, we first need to know whether a particular pathogen can cause disease on a particular plant species (pathogenicity). If it can, then we know that there are biotypes, races, or strains within the pathogen species that vary in their ability to cause disease on that host plant (virulence). Virulence varies both in terms of **which** cultivars can be infected and in the **severity** of disease that may occur. Pathogenicity and both the qualitative and quantitative characteristics of virulence are governed by genetic interactions between plants and pathogens.

Gene-for-Gene Interactions

Gene-for-Gene Interactions

- **effectors:** pathogen molecules, usually proteins, that are translocated into host cells, where they may act to directly manipulate host innate immunity
- **elicitors/effectors** from the pathogen: avirulence (*avr*) genes
- **receptors** in the plant: resistance (R) genes
- *avr* + R = hypersensitive response (HR)

Plant pathogens face many challenges, including survival in the absence of a host plant, coordination of their activities with those of their hosts in time and space, and establishment of an infection in a plant. From the previous section, it is clear that plants anticipate the possibility of invasion with both constitutive and induced structural and chemical defenses. Plants respond to invasion with systemic signals that can turn on defenses in distant tissues and new growth and that are effective against a variety of pathogens (i.e., SAR). What is the relationship between these defenses and the genetic interactions between a host plant and its pathogens?

Immunity in plants is the most common interaction with microorganisms, both for the many microorganisms that are never plant pathogens and even for those that *are* plant pathogens. Host range studies confirm this principle. The genetic basis of the interaction between hosts and pathogens has been known for many years.

H. H. Flor advanced our understanding of this interaction. He was the first to study the inheritance of resistance in a host (flax, *Linum utilissium*) at the same time that he studied inheritance of virulence in the pathogen (the flax rust fungus, *Melampsora lini*). From his studies, the term **gene-for-gene interactions** was created to help explain the dynamic genetic interactions between plants and their pathogens. Gene-forgene interactions have been found to apply to many, but not all, host-pathogen interactions, including those involving bacteria, fungi, nematodes, viruses, and even some insects.

► **DID YOU KNOW?**

In 1931, H. H. Flor began his work at the North Dakota Agricultural College in Fargo and worked there for the rest of his career. He selfed and crossed many races of *Melampsora lini*, the flax rust fungus, and, by applying them to the plant, found many resistance genes in flax. It took six years before he first published this work. Flor was the first plant pathologist to study the inheritance of resistance in the host and virulence in the pathogen. This allowed him to deduce what is popularly known as the gene-for-gene hypothesis.

The modern interpretation of what Flor first recognized is that resistant plants have **dominant resistance genes (R genes)** that match **dominant avirulence genes (*avr* genes)** in the pathogen. Proteins encoded by *avr* genes are called elicitors or the newer and more inclusive term, **effectors**. Effectors are pathogen molecules, usually proteins, that are translocated into host cells, where they may act to directly manipulate the innate immunity of the host. The majority of *avr* genes encode enzymatic proteins that target plant proteins during disease development. This enzymatic activity promotes disease development in susceptible host plants but can be recognized by a corresponding receptor in resistant plants. These receptors are coded for by R genes.

		Plant receptor	
		+	**–**
Pathogen elicitor	+	**R**	**S**
	–	**S**	**S**

The **incompatible reaction** of **R gene receptors** in the plant and ***avr* gene elicitors** in the pathogen leads to resistance (R). The hypersensitive response (HR) triggers the rapid accumulation of defense compounds. If the plant lacks receptors or the pathogen lacks elicitors, the plant will be susceptible (S) to that pathogen, and disease will develop.

A key feature of R-gene resistance is the development of a **hypersensitive response (HR)**, the production of a tiny area of necrosis—one or a few cells—in response to infection (Figure 9.33). This localized cell death is a plant reaction that occurs quickly and only in a limited area when the plant recognizes that it has been invaded, unlike the more extensive blighting and other general necrosis in many plant diseases. The HR seems to signal the induction of a cascade of the induced defenses described in Section B. Much remains to be learned about the details of HR, but it appears to have a genetic component.

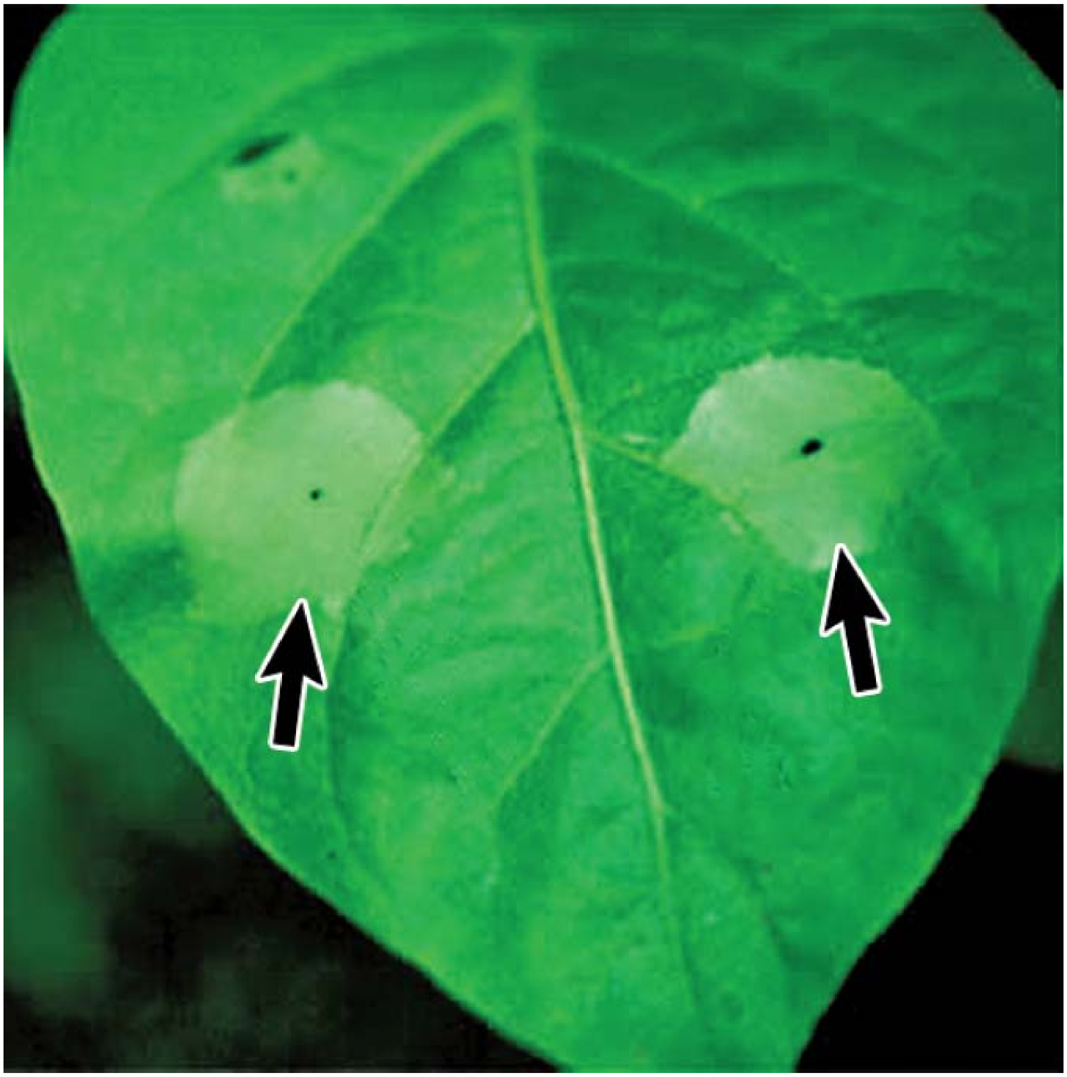


Figure 9.33. Areas (arrows) of hypersensitive response (HR) in pepper in response to infection by a bacterial spot pathogen.

R-gene resistance gives many host-pathogen interactions their genetic specificity. The **incompatible reaction** resulting from the recognition of ***avr* gene effectors** from the pathogen by **R-gene receptors** in the plant leads to resistance. The *avr* genes were first discovered in the bacterium *Pseudomonas syringae* and soon after in the fungus *Fulvia fulva* (syn. *Cladosporium fulvum*). Bacterial *avr* genes from plant pathogens share features with genes that have a similar function in animal pathogens.

Bacterial *avr* gene effectors are delivered into the host cell by a specialized system called a type III secretion system (TTSS). The

TTSS is a macromolecular assembly consisting of over 20 proteins and has been described as analogous to a syringe. Key structural TTSS proteins are encoded by **hypersensitive response** and **pathogenicity (*hrp*** or “harp”) **genes** in bacteria. ***Hrp* genes** are required to elicit resistance in resistant plants and trigger disease in susceptible plants. They have been found in nearly all gram-negative bacteria. The reason for this seemingly contradictory function of *hrp* genes in both resistant and susceptible reactions is that *hrp* genes encode components of the TTSS. Thus, the outcome of the bacteria-plant interaction depends on how specific receptors in the plant recognize the various bacterial effectors secreted into the host cell. In 1992, one of the elicitor proteins of a *hrp* gene was identified and named harpin; it is now produced as the commercial product Messenger used to trigger SAR in plants.

hrp Genes in Bacteria

hrp = hypersensitive response and pathogenesis
- elicit HR in resistant plants
- trigger disease in susceptible plants

The first plant R gene to be cloned and molecularly characterized was in corn in 1992. It codes for an enzyme that inactivates the host-specific toxin produced by race 1 of *Cochliobolus carbonum*, the causal agent of northern corn leaf spot disease. Because this toxin is an essential pathogenicity factor, its inactivation leads to resistance in the plant. This is an example of resistance that involves the gene-forgene interaction but does not involve the hypersensitive response (HR).

Other plant R genes have been identified and cloned in recent years. An R gene from a resistant tomato cultivar was cloned and transferred by genetic engineering to a susceptible tomato cultivar, making that cultivar resistant to a bacterial disease. Even though a number of plant R genes have been identified, their function in most cases is still unknown. New research will no doubt aid in our understanding of the critical first moments of recognition between hosts and pathogens. In the meantime, plant breeders and pathologists are working together to select plants with disease resistance.

Many host-pathogen interactions can be described by the gene-for-gene interaction—the matching of resistance (R) genes for plant receptors with avirulence (*avr*) genes for pathogen elicitors, leading to resistance. Some bacteria have *hrp* genes, which cause a hypersensitive response in the resistant plant and pathogenicity in the susceptible plant. Research in molecular biology is leading to an understanding of the events that result in disease resistance.

How can we create disease-resistant plants?

People create conditions that favor disease epidemics. We tend to plant monocultures of genetically uniform plants (Figure 9.34). This can be seen in greenhouses, field crops, lawns and golf courses, and forest plantations around the world. In nature, if an epidemic occurs in a plant population, only the survivors will grow the following year. Over time, resistance to a pathogen will increase. In agriculture, we often replant susceptible plants repeatedly, using a variety of other disease management practices to minimize disease. The dynamic genetic interactions that help keep a balance between hosts and pathogens in natural ecosystems are disrupted in monoculture agroecosystems.

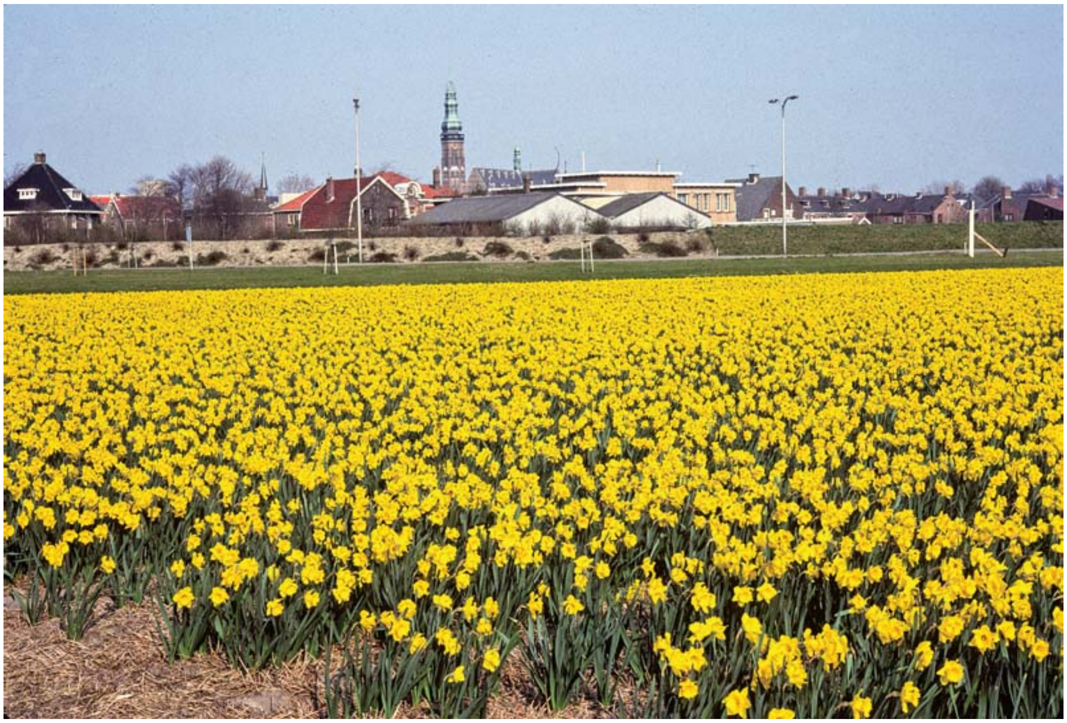


Figure 9.34. Field of genetically uniform daffodils. Gardeners prefer uniform flowers, but these plants are all equally susceptible to potential pathogens.

> Optimal yield may depend on breeding varieties in which the appropriate compromise is found between the demands for uniformity and the advantages of diversity.
>
> —Peter Day, 1974

Genetic Variation in Plants and Their Pathogens

Both hosts and pathogens have sources of genetic variation that play a role in genetic host-pathogen interactions and in our ability to create disease-resistant plants. Host plants, parasitic plants, nematodes, and fungi are all eukaryotes. They have nuclear genes that undergo **genetic recombination** during normal sexual cycles. In addition, some genes are associated with organelles in the cytoplasm, such as mitochondria and chloroplasts, and are passed on to the next generation in these structures. Such **cytoplasmic inheritance** (also called extrachromosomal or maternal inheritance) can be important in disease resistance.

Sources of Genetic Change in Plants and Pathogens

	Sexual cycle	Cytoplasmic inheritance	Mutation
plants	X	X	X
fungi	X	X	X
nematodes	X	X	X
bacteria			X
viruses			X

Note: Fungi and bacteria each have some additional specialized means of genetic variation.

Fungi have additional sources of genetic variation. Through **heterokaryosis**, genetically different nuclei may join after fusion of two mycelia. Laboratory studies of asexual stages of ascomycetes have demonstrated genetic recombination in the absence of typical sexual structures through a process known as **parasexualism**, but the importance of this process in nature is uncertain.

Mutation is a source of genetic variation in all organisms. Mutation can modify both nuclear and cytoplasmic genes of eukaryotes. Mutation is the most significant source of genetic change in bacteria. Bacteria also may absorb foreign DNA from their environment, exchange plasmid genes, and transfer genes via viruses (bacteriophages), although these events occur less often than mutations. Mutation also is the principle source of genetic variation in viruses, whether their genome is composed of DNA or RNA.

In general, pathogens probably have the advantage over plants in terms of genetic variation because of their shorter life cycles and their ability to reproduce quickly and in large numbers. It is a continuing challenge to select plants with effective and stable genetic resistance to important pathogens.

Plants and their pathogens both have various mechanisms of genetic variation that lead to a dynamic genetic interaction in natural ecosystems. In agroecosystems, we usually control the genetics of the plants, which may put them at a genetic disadvantage relative to the genetic change that can occur over the short generation times of many kinds of pathogens. Sources of genetic variation in eukaryotic organisms (e.g., plants, fungi, nematodes) are the sexual cycle and the mutation of both nuclear and cytoplasmic genes. Mutation is the most important source of genetic variation in bacteria and viruses.

Specific (R Gene) Resistance

Even before the structure of DNA was discovered (in 1953), disease-resistant plants were developed by back-crossing plants with dominant R genes with plants having desirable horticultural characteristics. The resistance conferred by R genes is **qualitative** (+ or –) and complete against a specific race of the pathogen (Figures 9.35–9.37). R genes were not available for many crops because of their limited genetic diversity. Thus, it was often necessary to travel to the **center of origin** (the native home) of the host plants, where genetic diversity was greatest, to find

germplasm for resistance breeding. Wild relatives and undomesticated versions of crop species as well as **land races**, which are selected by indigenous farmers, continue to be important sources of R genes for breeders.

Figure 9.35. Variation in resistance to dollar spot among cultivars of fine fescues. The demand for uniformity in turfgrass growth characteristics also results in uniform disease resistance or susceptibility.

Factors That Affect R Gene Durability

1. Is the disease monocyclic or polycyclic?

Each pathogen generation increases the opportunity for multiplication of the new race (biotype or strain).

2. Does the pathogen have significant sources of genetic variation?

Sexual reproduction produces genetic recombination with every generation. Mutation is more significant in organisms with shorter generation times.

3. Is the pathogen airborne or soilborne?

New races of airborne pathogens are distributed more quickly.

4. Is the environment conducive to disease?

New races multiply more slowly in a year in which moisture and/or temperature is not optimal.

5. Are other disease management methods available for the disease?

If plants are protected by other means, such as fungicide applications, crop rotation, or minimizing leaf wetness, there is less selection pressure for new pathogen races to increase.

Figure 9.36. Beans resistant (left) and susceptible (right) to the prevalent race of rust caused by *Uromyces appendiculatus*.

Figure 9.37. Rice susceptible (left) and resistant (right) to tungro virus disease.

► DID YOU KNOW?

Land races are plant cultivars that have been selected by indigenous farmers over many generations to meet the needs of their local areas. Studies have shown that the greatest number of resistance genes is usually found in land races where the host and pathogen have coexisted for long periods. For example, resistance genes to most of the major barley diseases have been found in land races selected by farmers in Ethiopia, where barley originated.

Some of the early scientists incorrectly believed that their disease-resistant plants would never again be vulnerable to epidemics of that disease. They did not anticipate that, following the gene-for-gene hypothesis, different races (or biotypes or strains) exist in most pathogen populations. Soon after a new resistant cultivar is widely planted, selection pressure leads to an increase of pathogen races that can overcome the resistance. The formerly resistant plants are susceptible to the new pathogen races. The resistance was said to have “broken down,” but, in fact, the genetic change was not in the plant; the pathogen race that could infect the resistant cultivar had “overcome” the R gene.

The basis of **specific resistance** is usually **monogenic (single-gene resistance)** or **oligogenic** (involving a few genes) and is sometimes called **vertical resistance**. It usually involves dominant

R genes that interact with dominant *avr* genes in the pathogen. The R genes usually have a large effect on resistance against specific races of the pathogen. However, they will be effective only if those races are the predominant ones in the pathogen population. How quickly a resistant cultivar might fail depends on several factors (see box).

Over time, the failure of specific resistance became known as the **boom-and-bust cycle** of plant breeding. First, a breeder produces a cultivar with specific resistance. If the cultivar is popular, it is more likely to be widely planted, causing greater selection pressure on the pathogen to overcome the R gene. New races that can overcome the R gene become predominant, leading to widespread failure of the once-resistant cultivar. On the other hand, some R genes have remained effective for many years. These tend to be those used for **monocyclic**, soilborne diseases such as Fusarium wilt, Verticillium wilt, and root-knot nematode or cyst nematode diseases.

Boom-and-Bust Cycle of Resistance Breeding

1. New cultivar with R gene is released.
2. Cultivar attributes make it popular, and it is widely planted.
3. Selection pressure is placed on the pathogen to overcome the R gene.
4. New pathogen race infects cultivar with R gene.
5. The cultivar is no longer disease-free.

Boom-and-Bust Examples

Late blight (*Phytophthora infestans*) of potatoes in North America

Mid-1920s: Potatoes with resistance gene (R1) from wild *Solanum demissum* developed
Mid-1930s: Race 1 of *P. infestans* appears and infects potatoes with the R1 gene (and those without it)
Mid-1960s: Nine R genes have been identified and overcome by new races of the pathogen

Stripe rust (*Puccinia striiformis*) of wheat in Europe
Early 1980s: Popular winter wheat cultivars developed:
  Slejpner, with resistance gene *Yr9*
  Hornet, with resistance gene *Yr6*
1986: 90% of rust population has virulence gene *Yv9* to overcome *Yr9*
1989: Second pathogen race appears with *Yv6* and *Yv9* virulence genes, resulting in severe stripe rust epidemics in both Slejpner and Hornet wheat cultivars

Many devastating epidemics of rusts of cereals and late blight of potato were the result of the failure of specific R gene resistance. These epidemics are important examples of resistance failures. These epidemics became significant because the pathogens are airborne, the diseases are **polycyclic**, and the crops cover large geographical areas, especially in the case of cereal crops. Studies of specific R genes in wheat suggest that as many as 56,000 races of *Puccinia graminis* f. sp. *tritici* (stem rust) might exist, posing a significant challenge to breeders.

In 1918, just before the barberry eradication program began, it was possible to find as many as 28 races of the stem rust pathogen in the wheat fields of North America. The removal of the **alternate host**, barberry (Figure 9.38), broke the sexual cycle of the fungus and left it with only urediniospores that are blown north by wind via the Puccinia pathway each year from overwintering sites in the south. Barberry eradication delayed the epidemics, reduced inoculum, and provided only asexual spores to initiate epidemics. Without its sexual cycle, *P. graminis* had only mutation as a source of genetic variation. Today, it is common to find only five races of *P. graminis* in wheat fields, and only two races predominate. The reduction in genetic variability of the stem rust pathogen results in a longer-lasting resistance in cultivars developed by wheat breeders. “Rust gardens” of wheat lines with single R genes are planted each year to help monitor the pathogen races and predict which R genes should be deployed for the coming year.

Figure 9.38. Spraying common barberry with herbicide to eliminate the alternate host of *Puccinia graminis*. This reduces the genetic variation in this important rust fungus by eliminating the sexual cycle.

The usefulness of R genes can be prolonged by activities that reduce the pathogen population. This simply reduces the probability that a new race will become dominant. Cultural practices that help to minimize disease, such as timing irrigation to reduce leaf wetness, cleaning up infested debris, and crop rotation, are all means to reduce pathogen populations and slow genetic variation.

The Vertifolia Effect

This phenomenon describes the inadvertent loss of general resistance when R genes are introduced into cultivars. Vertifolia is a potato cultivar with specific (R gene) resistance to late blight. With late blight under control, Vertifolia was used in additional breeding cycles for the selection of other genetic improvements. When a new race of *Phytophthora infestans* overcame the R gene, the Vertifolia potatoes were found to have lost much of the general resistance they had before the R gene was added.

General (Race-Nonspecific) Resistance

As breeders and pathologists became aware of the limitations of specific resistance, especially for airborne pathogens and polycyclic diseases, they began to search for longer-lasting forms of genetic resistance. Rather than searching for single dominant R genes that protected against a single race of a pathogen, they began to try to breed plants with resistance against all pathogen races. This is a **quantitative** form of resistance. This is observed in nature, where individual plants vary in their levels of resistance, but some disease still occurs. If a disease is described as a minor disease of a plant, it is likely that the plant has good general resistance to that pathogen. Despite the term *general*, keep in mind that this is resistance to a single disease and is not effective against other diseases.

General Resistance

- often controlled by many genes (multigenic or polygenic)
- also called horizontal resistance, race-nonspecific resistance, or durable resistance
- usually partially effective against all races of a pathogen
- subject to environmental influence

General resistance is often **polygenic** (or **multigenic**) and is also called **horizontal resistance** or **race-nonspecific resistance**. It is conferred by a number of genes, each of which has some influence on the pathogen. Plants with general resistance usually have only **partial resistance**, in which disease development is slower, lesions may be smaller, and production of secondary inoculum is reduced (Figures 9.39 and 9.40). For example, general resistance to rust diseases in some cereals is called “slow rusting.”

Figure 9.39. Potato leaf from a susceptible plant (right) and leaves from plants with varying levels of general resistance. General resistance functions against all races of the late blight pathogen, *Phytophthora infestans*. In resistant plants, lesions are smaller, develop more slowly, and produce fewer sporangia.

Figure 9.40. Potatoes with general resistance to late blight. Note dead plants of a commercial cultivar in the foreground.

A great advantage of general resistance is that it is considered stable; it is sometimes called **durable resistance**. Although a pathogen can potentially overcome the effects of a single R gene, the probability of overcoming multiple genes decreases with the presence of each additional gene. On the other hand, it is more difficult to select plants with general resistance because it does not involve single genes with major effects. General resistance is accumulated over time using populations of plants. Genetic engineering may make the development of plants with polygenic resistance easier (see below).

Successful General Resistance: Common Corn Rust

Common corn rust (*Puccinia sorghi*) is not a serious problem for most growers in the United States, even though the disease can be found in most years. Two kinds of general resistance to common rust have been identified in corn. One form makes mature leaves less susceptible to infection. The other form reduces the number and size of the uredinia or increases the latent period. This restricts the disease sufficiently in mild climates but may be ineffective in tropical climates.

Environmental factors may modify the effectiveness of general resistance. General resistance may not perform as well under high disease pressure due to favorable weather conditions or high levels of inoculum. Thus, cultural practices that help minimize disease and favor plant growth will enhance the effectiveness of general resistance. Alternatively, practices that favor disease development can make resistant plants perform poorly.

Because specific resistance is much easier to select, it is still a popular form of resistance. The successful use of genetic resistance and the deployment of R genes within the framework of plant disease epidemiology are discussed further in Chapter 11.

Ug99 Threatens Global Wheat Production

Scientists have estimated that Ug99, a new race of the wheat stem rust pathogen, could potentially reduce world wheat production by 60 million tons. Stem rust can cause 50–70% yield losses and even total destruction, depending on the stage of plant development when the disease becomes severe. Resistance to stem rust is based mainly on gene *Sr31*, but Ug99 can overcome this resistance gene. Ug99 was first detected in Uganda in 1999. It has spread from East Africa to Yemen and Sudan and was identified in Iran in 2008. The Borlaug Global Rust Initiative has as its goals developing resistant cultivars, providing clean seed, and upgrading breeding and plant-production services in response to this threat. The website Pathotype Tracker—Where is Ug99? monitors the situation. Another race of *Puccinia graminis* var. *tritici* that can overcome additional resistance genes has recently emerged in Kenya.

Specific (R gene) resistance is relatively easy to manipulate, but it may fail when a new pathogen race increases (the boom-and-bust cycle of plant breeding). Specific resistance may be very effective for soilborne pathogens and monocyclic diseases. It will be more durable when accompanied by other methods that limit disease development. General resistance is often more stable (durable) and is effective against all races of a pathogen. This polygenic type of resistance is usually only partial and can be enhanced by additional management practices.

Gene Silencing Using RNA Interference (RNAi)

RNA interference (RNAi) takes advantage of natural cell activities that regulate the abundance of mRNAs and defend plants from virus infection. In early experiments, scientists tried to block mRNA by inserting complementary single-stranded RNA or DNA into cells. They hoped that if the mRNA were blocked or destroyed, the gene from which it was transcribed would no longer be functional, a process called **gene silencing**. It was later discovered that double-stranded RNA (dsRNA) is needed to successfully block the mRNA and thereby silence the gene. Although RNAi was first discovered in plants, the 2006 Nobel Prize was awarded to scientists studying how to silence a gene in the bacteria-feeding nematode, *Caenorhabditis elegans*. In some of their experiments, the nematodes were soaked in a solution of dsRNA that was complementary to the sequence of the gene to be disabled. In other experiments, the nematodes were fed on bacteria that had been genetically transformed to express the dsRNA.

In RNA interference, the dsRNA is processed by a protein called Dicer, which breaks it up into tiny fragments. Helicase activity unwinds the dsRNA pieces to generate the small interfering RNAs (siRNA), which are single-stranded. The siRNA is incorporated into a protein complex called RISC (RNA induced silencing complex), in which it is able to bind to mRNA, destroy it, and effectively block the function of, or silence, the gene from which it was transcribed. In genomics research, the ability to block the function of a gene by targeting its mRNA for destruction with RNAi can help identify the gene's role in an organism. Key pathogen genes can be identified and possibly blocked. Recently, RNAi was able to silence an important parasitism gene in root-knot nematodes, resulting in a dramatic decrease in nematode infection. The dsRNA was delivered from a transgenic plant (see next section), suggesting a novel way to develop resistance to plant pathogens in crops. Virus-induced gene silencing has become widely used to assign functions to host plant genes. As the genome sequences of many pathogens become available, gene silencing offers exciting new approaches to plant disease research.

RNA interference (RNAi) uses double-stranded RNA complementary to a specific mRNA to block or silence a gene by targeting its mRNA for destruction. RNAi potentially can be used to block or silence important pathogen genes for plant protection.

Biotechnology and Genetic Engineering for Disease Resistance

Genetic engineering is the transfer of specific genes between organisms using enzymes and laboratory techniques rather than traditional biological crossing. It is based on the concept that all living organisms share the same genetic code. Theoretically, any "gene" should be able to function in any organism. Clearly, the situation is not that simple. Even after genes are identified, cloned, and added to the genome of a new plant, the genes may be modified by their location on a chromosome, by the presence of other genes, and by the environment in which the organism lives. However, genetic engineering is a tool that overcomes many limitations of natural reproductive systems.

USE OF GENETIC ENGINEERING FOR DISEASE RESISTANCE

Advances in molecular biology have improved our ability to identify and manipulate R genes. As more R genes are identified and their manipulation becomes more efficient, some scientists hope that this may offer a new approach to routinely creating reliably resistant plants. A particular advantage of genetic engineering over traditional breeding is that genes potentially become available from any organism, not just from plants of the same species, as is the case with conventional breeding. In many cases, even closely related wild relatives of economically important plants remain genetically isolated by pollen incompatibilities, frustrating traditional breeding efforts. Genetic engineering allows access to these genes for plant improvement.

Because virus diseases are particularly difficult to manage, there has been considerable interest in finding new approaches to their management. In some cases, selection of R genes through traditional breeding has been successful. For a few virus disease problems, high value crops (e.g., citrus) are inoculated with a mild strain of a virus (e.g., *Citrus tristeza virus*) to protect them against infection by more severe strains of the virus. The exact mechanism for this **cross-protection** is being studied.

► DID YOU KNOW?

> Cross-protection was first demonstrated by H. H. McKinney in 1929. He isolated a new yellow mosaic strain of *Tobacco mosaic virus* (TMV) from tobacco plants in the Canary Islands. TMV usually causes a green mosaic. He inoculated plants infected with the green mosaic strain with the new yellow mosaic strain, but they never developed the yellow mosaic symptoms. This demonstrated cross-protection by the green mosaic strain of TMV. Later studies showed that cross-protection is successful with different strains of the same virus or with closely related viruses, but those plants remain susceptible to other viruses.

Disadvantages of Cross-Protection Using Mild Virus Strains

- potential for mutation to a severe strain
- potential synergistic reaction with other viruses
- source of inoculum for other plants that might be more susceptible

There is concern that the mild virus strains might mutate or serve as inoculum reservoirs for infection of different host plants that might develop more severe symptoms. Also, viruses that usually cause mild symptoms in a plant sometimes cause severe symptoms when the plant is infected by a second virus. (This is an example of synergism.) For these reasons, many growers have been uneasy about this technique, but they have used it because there was no alternative way to protect the high value perennial plants.

To avoid the use of actual virus infection, a different approach was developed in which the coat protein gene of *Tobacco mosaic virus* (TMV) was inserted into the genome of tomato plants, using genetic engineering. These modified plants became resistant to

TMV. This process is called coat protein-mediated resistance. Since this initial work was performed, coat protein genes of a number of other viruses, along with some other virus genes, have been cloned and used to produce virus-resistant plants (Figure 9.41).

Figure 9.41. Transgenic papaya resistant to *Papaya ringspot virus* (PRSV) (right) and susceptible papaya (left). Plant embryos received the coat protein gene of PRSV by biolistic transformation.

The most common way to insert a new gene into a plant is through the use of the bacterium *Agrobacterium tumefaciens*, the natural genetic engineer. In its role as a plant pathogen, genes are transferred from the Ti (tumor-inducing) plasmid of the bacterium into the genome of a host plant. With the use of recombinant DNA techniques, the plasmid is “disarmed” so that the genes responsible for disease are removed and are replaced with the genes that scientists wish to add to the plant’s genome. This works well for a wide variety of plants. The technique requires that the plants be regenerated from a single cell because each cell of the engineered plant must contain the new gene.

Sometimes the Ti plasmid cannot be used to transform plants. A common alternative is **biolistic transformation** using the “gene gun,” which bombards **protoplasts** (plant cells from which the cell wall has been removed) with metal particles (often tungsten) coated with the foreign genes (Figure 9.42). The gene gun is much less efficient than the Ti plasmid for gene transfer, and the results are less predictable genetically. In both cases, it is necessary to harvest seeds from the **transgenic** plant to determine that the new gene has actually become a functional part of the genome and is able to be transferred through seed to new generations.

Genetic engineering: transfer of specific genes between organisms using enzymes and laboratory techniques rather than traditional biological crosses; in plants, usually accomplished using the Ti plasmid of *Agrobacterium tumefaciens* or the "gene gun" (biolistic transformation)

Transgenic: containing a gene from a different species; used to describe the products of genetic engineering

Genetically modified organism (GMO): a transgenic organism

Biotechnology: use of laboratory techniques for multiplication or genetic modification of organisms; includes tissue culture and cell culture

Figure 9.42. The "gene gun" used for biolistic transformation of plants.

Uses of Protoplasts for Plant Improvement

- rapid screening for resistance to toxins and disease factors
- protoplast fusion provides genetic variation in infertile plants and in asexual fungi; the latter is useful for improving biological control agents
- generation of haploid plants to expose recessive genes
- reduction in chromosome number to improve genetic manipulation of polyploid plants

Genetic engineering offers many new approaches to disease resistance. Some obvious approaches are to add genes for additional receptors that can recognize pathogen invasion or to enhance the passive or active defenses a plant already has. Genes from other organisms might give a plant defenses that it never had before. As we better understand exactly how pathogenesis occurs, we can try to interfere with how pathogens find their hosts or inactivate important pathogen enzymes or toxins. Recent studies of the early molecular events that occur in host-pathogen interactions will open avenues of plant protection that we cannot even imagine now.

In addition to genetic engineering, **biotechnology** has some other applications in the production of improved plants. For example, it is sometimes much easier to manipulate protoplasts and then regenerate whole plants from them rather than working with whole plants over many generations (Figure 9.43). Protoplasts have been used to more rapidly select plants with resistance to pathogen toxins and other specific disease factors. Also, some plants, such as many bananas, are infertile and cannot be crossed using traditional breeding techniques. **Protoplast fusion** and regeneration of plants from the fused protoplasts provides a way to create genetic variation for selection.

Figure 9.43. Protoplast of a plant cell from which the cell wall has been removed enzymatically, leaving only the cell membrane to enclose the cell contents.

Through biotechnology, plants can be generated from pollen and ovule cells that have half the chromosome number of the parent plant. Commonly, this produces haploid plants and exposes the presence of recessive genes that may be desirable in a breeding program. In polyploid plants, the chromosome number can be reduced to more manageable levels.

Biotechnology is helpful even in traditional breeding programs. Molecular biology techniques can identify R genes and their locations on plant chromosomes. **Marker-assisted selection** uses genetic markers that are easy to identify and that are linked to the R genes. This allows the DNA of progeny to be rapidly screened for the presence of R genes without growing the plants to maturity to observe their reaction to disease (see box).

Marker-Assisted Selection Helps to Identify:

- resistant plants even before a pathogen has invaded a particular geographical area
- resistance genes in plants with long life cycles, such as trees
- resistance to pathogens that require a long time for development
- resistance to pathogens that require vectors or special environmental conditions

CONCERNS ABOUT GENETIC ENGINEERING AND BIOTECHNOLOGY

Regulation of GMO Crops

Institutional Biosafety Committee
regulates any potentially hazardous biological research, including genetic engineering

Animal and Plant Health Inspection Service (APHIS) of the USDA
regulates planting, transport, and import of GMO plants; determines whether they are likely to have negative agricultural or environmental effects

Food and Drug Administration
regulates food safety; determines tests that must be completed for GMO foods, such as tests for possible allergens, known toxicants, altered nutrient levels, new substances, or antibiotic resistance markers

Environmental Protection Agency (EPA)
regulates GMO crops that contain a plant protectant such as a *Bacillus thuringiensis* (*Bt*) gene for insect resistance or a virus resistance gene; determines how the protection works, reviews environmental effects, and may require a resistance management plan. If the mechanism of resistance results in the production of a toxin, the EPA sets the tolerance (legal residue) level.

State regulations
may more closely regulate GMO crops than federal laws do

International treaties and trade agreements
may regulate GMO crops

The use of biotechnology, and genetic engineering in particular, is controversial. Plant pathologists are well aware of the dangers of introducing "new" organisms into ecosystems and the potential dangers of widespread use of a single genetic factor. These dangers exist in traditional breeding programs, as well as with genetic manipulation accomplished in the laboratory. Plants and pathogens that have been genetically modified through laboratory techniques are often called **genetically modified organisms** (**GMOs**). Concerns about GMOs generally fall into three areas:

1. **Ecological concerns**. New gene combinations may lead to surprising results. Scientists must evaluate invasiveness of the GMOs; unintended effects on plant growth, quality, and yield; and the potential for genes to escape to closely related wild relatives.
2. **Safety concerns**. New gene combinations must be evaluated for toxicity and allergenic potential for humans and domesticated animals that ingest harvested plant parts, as well as for potential toxicity or other negative effects on nontarget organisms. Although genes inserted through genetic

engineering are usually very well studied, they may cause the production of newly formed substances and altered nutrient levels.

3. **Economic concerns**. Although difficult to evaluate, the use of GMOs impacts the survival and use of land races of major crops and other traditional crops, especially in developing countries. Issues of ownership of genes by corporations and access to genetic resources, most of which are in the tropical areas of the world, are complex. Oversight or stewardship of genes that impart resistance to insects and pathogens varies from country to country, but it has worldwide impact. The boom-and-bust cycle is all too familiar to plant pathologists, but companies selling seed of plants with R genes may want to quickly maximize profits to cover the high costs of product development. We must be careful not to squander our genetic resources.

Although the mass media focus on the issues related to GMOs, it is important to realize that most of these same issues apply also to plants from traditional breeding programs. For example, potato tubers from traditional breeding programs must be tested to make sure that they do not have poisonous levels of the natural alkaloids found in potato leaves. Effects on plant growth and development, possible increases in potential natural toxins, and stewardship of R genes must be evaluated for all new gene combinations, including both GMOs and plants from traditional breeding programs. The production of safe and plentiful food and fiber depends on continual improvements and careful evaluation before use.

► **DID YOU KNOW?**

The National Center for Genetic Resources Preservation is in Fort Collins, Colorado. In addition to being a seed bank, NCGRP is a repository for animal genetic resources in the form of semen and plant genetic resources in the form of graftable buds or *in vitro* plantlets.

Biotechnology and genetic engineering offer efficient means to manipulate genes from plants and other organisms. Most plants are transformed using the *Agrobacterium* Ti plasmid or biolistic transformation (the gene gun). Protoplasts can sometimes be used to manipulate and screen germplasm. Marker-assisted selection may speed the selection of resistant plants. New gene combinations, whether through biotechnology or traditional breeding, should be evaluated for ecological and economic impacts as well as for food safety.

Optimizing Resistance to Pathogens

Genetic resistance to pathogens involves a combination of constitutive (preexisting) structural and biochemical defenses with induced structural and biochemical defenses. We are only beginning to understand the details of how a plant detects invading pathogens, turns on its defenses, and signals to distant plant parts that defenses should be induced. Only recently have SAR and ISR become weapons in the pathogen-fighting arsenal. How best to use them remains to be determined.

We already take advantage of general resistance as a means of defense against what are considered to be minor pathogens. We typically use combinations of specific resistance (R genes) and general resistance for defense against pathogens that pose a more significant risk to the health of a crop. The ability to identify R genes and other genes that can interfere with successful pathogen invasion and to manipulate them through genetic engineering offers a variety of efficient new approaches to the production of disease-resistant plants.

The gene-for-gene hypothesis describes the dynamic genetic interaction between plants and pathogens. It tells us that the fight against plant diseases will always exist. The National Center for Genetic Resources Preservation in Fort Collins, Colorado, is a central site for agricultural plant and animal genetic resources for the United States. Additional germplasm collections for specific crops are located in other areas of the United States. The Consultative Group on International Agricultural Research (CGIAR) is composed of agricultural research centers around the world, mostly in the major centers of crop biodiversity (Figure 9.44). These centers help maintain germplasm that is important to agriculture. In 2008, the Svalbard Global Seed Vault (SGSV) was established in Norway. The vault maintains seed collections drawn from CGIAR collections from Asia, Africa, Latin America, and the Middle East. It is located on a remote island near the Arctic Circle as an additional safeguard for these invaluable germplasm resources.

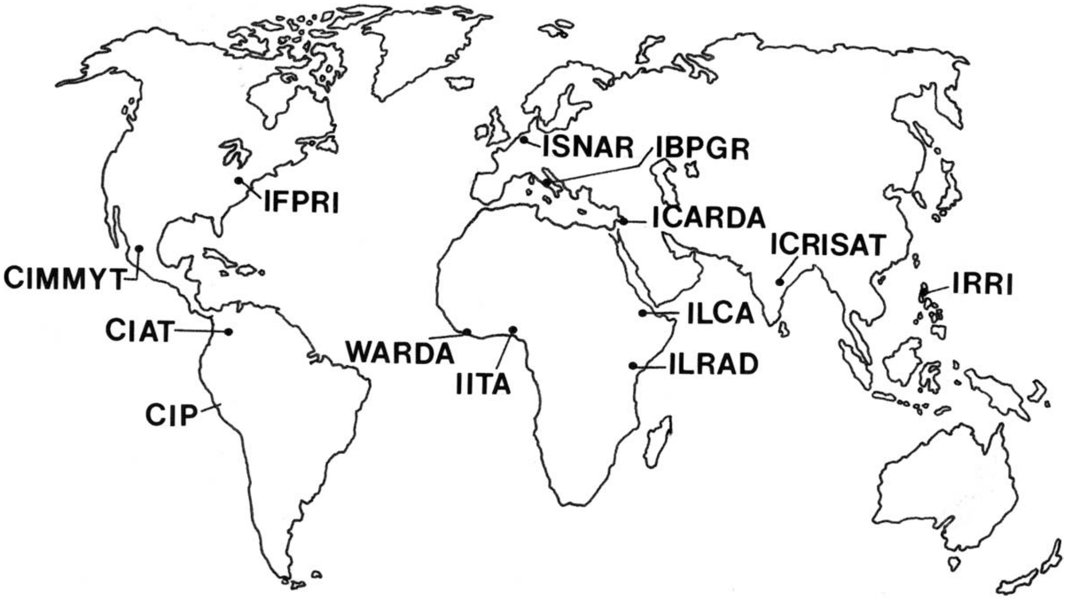


Figure 9.44. The international agricultural research centers.

► **DID YOU KNOW?**

The Consultative Group on International Agricultural Research (CGIAR) includes the research centers shown on the map (Figure 9.44). It also holds one of the world's largest collections of plant genetic resources in trust for the world community—over 500,000 accessions of more than 3,000 crop, forage, and agroforestry species. The collections include land races, improved cultivars, and the wild species from which those cultivars were created. The collections are under the auspices of the Food and Agriculture Organization of the United Nations. Germplasm in the in-trust collections is made available without restriction to researchers around the world with the understanding that no intellectual property protection is to be applied to the material.

Although these collections are an important resource for plant breeders, they are not a substitute for the biological diversity of natural ecosystems, especially in the centers of origin of our food and fiber crops (see Chapter 10). The loss of biological diversity is particularly acute in the tropical areas of the world, where many of the world's agricultural crops originated.

Part C. Genetic Interactions

Questions

1. What is the gene-for-gene interaction between plants and pathogens? How is it related to the boom-and-bust cycle of plant breeding for genetic resistance?
2. What are the sources of genetic variation in plants and each of the major pathogen groups?
3. How is specific resistance (R gene) to a pathogen different from nonspecific resistance (often polygenic) in plants? In what situations would you expect R gene resistance to fail most quickly? What are some ways to prolong the effectiveness of genetic resistance in plants?
4. Why is it inappropriate to say that a resistance gene has "broken down" when a resistant plant becomes susceptible again? What is a more appropriate way to describe this phenomenon?
5. What information about a pathogen and its host plant would be most useful when initiating a resistance breeding program?

Challenge Questions

1. Genetic engineering offers great promise for improvements in agriculture. What are some methods that are useful in the application of biotechnology to improve disease resistance in plants? Which of these applications would be difficult or impossible using traditional breeding methods?
2. Quarantine regulators (who govern the import of plant materials) and regulators of the release of GMOs (transgenic organisms) share many of the same concerns. Explain why this is true and discuss what you consider to be the most important factors to consider when determining the safe release of an

organism. Do you think that we need different regulations for the release of microorganisms and the release of plants?

3. Why are so many centers of crop diversity found in the tropics?

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

Genetic Interactions

alternate host avirulence (*avr*) gene biolistic transformation (syn. gene gun)
biotechnology
center of origin
cross-protection
dominant
durable resistance
effector
elicitor
escape
gene-for-gene interaction
gene silencing
general resistance
genetic engineering
genetically modified organism (GMO)
germplasm
heterokaryosis
horizontal resistance
hypersensitive response (HR)
hypersensitive response and pathogenicity (*hrp*) gene
immune
land race
monogenic resistance

marker-assisted selection
mollicutes
monocyclic
multigenic resistance
mutation
oligogenic resistance
parasexualism
partial resistance
pathogenicity
polycyclic
polygenic resistance
protoplast
qualitative resistance
quantitative resistance
race-nonspecific resistance
receptor
resistant
RNA interference (RNAi)
single-gene resistance
specific resistance
susceptible
tolerance
transgenic
vertical resistance
Vertifolia effect
virulence

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Supplementary Resources

Illustrated Glossary

Special Topic

An overview of plant defenses against pathogens and herbivores

Laboratory Exercises

Cytology of fungal infection

Following the disease progression of an ectotrophic root-infecting fungus

Exercise

Exercise 9.1. Choose three diseases from the Plant Disease Profiles in the APS Education Center. Try to find the following information about each:

a. How does the pathogen survive in the absence of the host plant?
b. How does the pathogen arrive at the plant?
c. How and where does the pathogen penetrate the plant?
d. Given the symptoms of the disease and information in the lesson, what chemical weapons does the pathogen use (enzymes, toxins, growth regulators) against the plant?
e. Does the pathogen exploit the new host by producing secondary inoculum?
f. Are resistant cultivars of the plant available?

Internet Resources

APS Resources

Cercosporin: A photoactivated toxin in plant disease
Introduction to population genetics of plant pathogens

Recommended Websites

Consultative Group on International Agricultural Research
Global Rust Initiative
Svalbard Global Seed Vault
Pathotype Tracker—Where Is Ug99?
The Unified Website for Biotechnology Regulation

Internet Research

Internet Research Exercise 9.1. Using the Internet, try to find information about a commercial "plant activator" that induces systemic acquired resistance (SAR) in a plant. Try searching on some commercial trade names such as Actigard, BioYield, Blockade, Elexa, or Messenger. How is the product used and for what kinds of plants is it recommended? What changes does the product induce in the plant? Should the product be considered a pesticide or a biological control agent?

Internet Research Exercise 9.2. Using the recommended Internet Resources and any others, find two examples of GMO plants with improved resistance to a disease. What ecological, safety, and economic concerns apply to these examples?

CHAPTER 10

How Do People Influence Plant Disease Epidemics?

IN THIS CHAPTER:

- **What is an epidemic?**
- **Can we predict plant diseases?**
- **How do people affect the susceptibility of plants to disease?**
- **How do people affect the environment of plants in ways that increase disease?**
- **How do people affect the ability of pathogens to cause disease?**
- **How do plant disease epidemics affect people?**

What is an epidemic?

When we begin to analyze a plant disease epidemic, we add a new component to the disease triangle. Disease can theoretically occur when the virulent pathogen and susceptible plant are together in a favorable environment. In reality, these factors must be combined over a period of time. So, our disease triangle should become a disease pyramid with a fourth vertex for time (Figure 10.1).

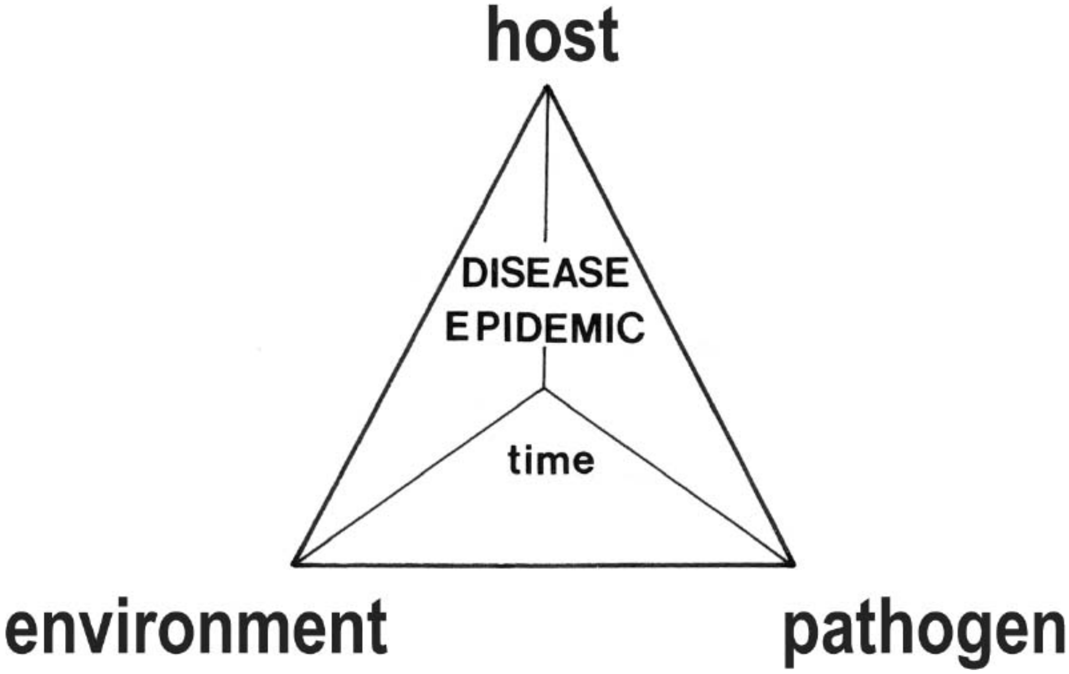


Figure 10.1. The plant disease pyramid.

Many people think of fast-developing and/or severe diseases as “epidemics.” Although these are the ones that grab our attention, any increase of disease with time is a concern to those interested in plant health. An **epidemic** is disease increase in a population over time. **Epidemiology** is the study of disease increases in populations, including the factors that influence their initiation, development, and spread. The more specific term **epiphytotic** is sometimes used to describe disease increase in a plant population, and **epizootic** describes disease increase in an animal population.

In the 1960s, J. E. Van der Plank developed two simple models for plant disease epidemics: one for monocyclic pathogens and one for polycyclic pathogens. **Monocyclic pathogens** complete only one generation during a single growing season. Using a banking analogy, these can be considered “simple interest” diseases. **Primary inoculum** (or **initial inoculum**) initiates disease, and no new inoculum is produced until the end of the growing season. The newly produced inoculum infects the next generation of plants. Many, but not all, monocyclic pathogens are soilborne. Examples include the pathogens that cause Fusarium and Verticillium wilts, clubroot of brassicas (crucifers), cedar-apple rust, corn smut, stinking smut of wheat (Figure 10.2), and many root diseases. In cooler climates, plant epidemics caused by nematodes are nearly monocyclic. Management of monocyclic pathogens focuses on **reducing the amount or the efficacy of the primary inoculum**.

Monocyclic Pathogens

- one generation of inoculum per season
- primary inoculum only
- often are soilborne pathogens

Management: reduce amount or efficacy of primary inoculum

Disease examples: cedar-apple rust, corn and cereal smuts, Fusarium and Verticillium wilts, diseases caused by nematodes in cooler climates

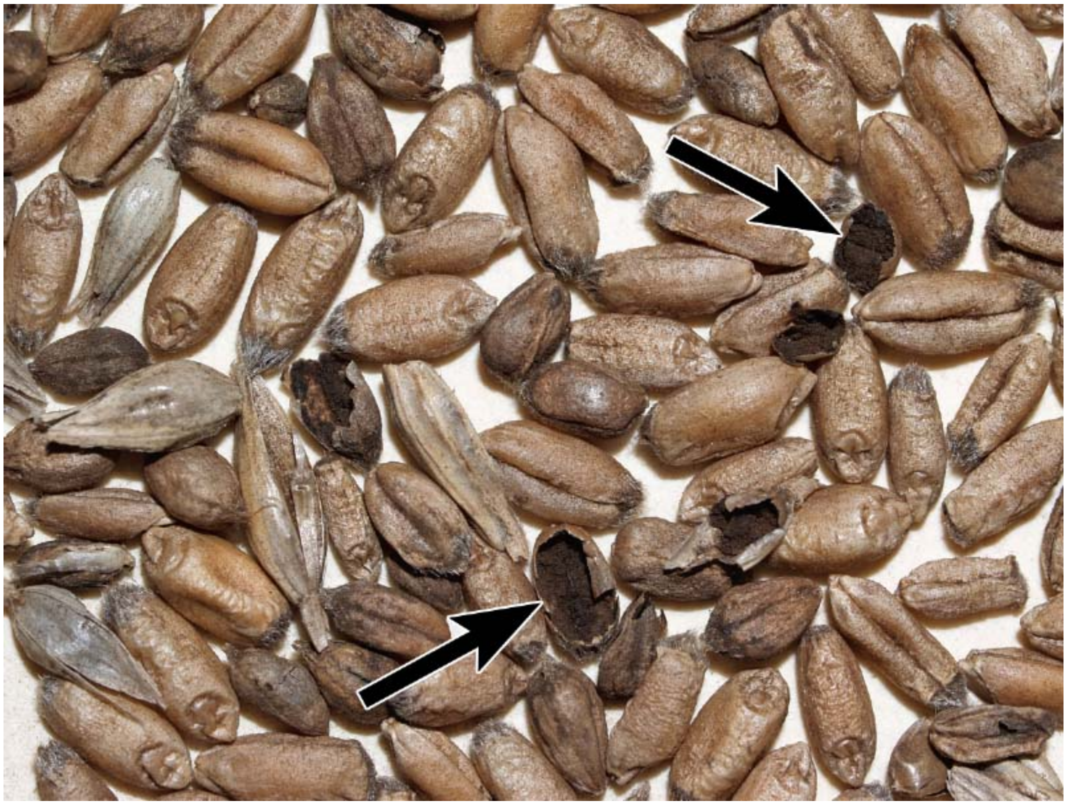


Figure 10.2. Masses of teliospores (arrows) from bunted wheat kernels with stinking smut, a monocyclic disease.

Polycyclic pathogens, in contrast, may complete several to many generations during the growing season. After the primary inoculum initiates disease, **secondary inoculum** is produced and causes additional infections. Infections may be on the same plant or on neighboring plants that are susceptible to the disease. Using a banking analogy again, these can be considered "compound interest" diseases.

Polycyclic Pathogens

- several to many generations of inoculum per year
- primary inoculum and secondary inoculum produced
- often are airborne or vectorborne

Management: reduce primary inoculum and reduce the rate of infection

Disease examples: apple scab, downy mildews, late blight, leaf spots, powdery mildews, rusts (most), virus diseases (most)

Although reducing the amount or efficacy of the primary inoculum is important, it may not be sufficient to manage the disease at an acceptable level. Even small amounts of primary inoculum of some pathogens can increase exponentially over several generations, resulting in explosive epidemics. It may be necessary to try to **reduce the rate of infection** for as long as inoculum remains a potential threat—often for the entire growing season. Diseases caused by polycyclic pathogens include anthracnoses, apple scab, downy mildews, many rusts (Figure 10.3), early blight of potato and tomato (Figure 10.4), late blight, leaf spots, powdery mildews, and many additional bacterial, fungal, and viral diseases.

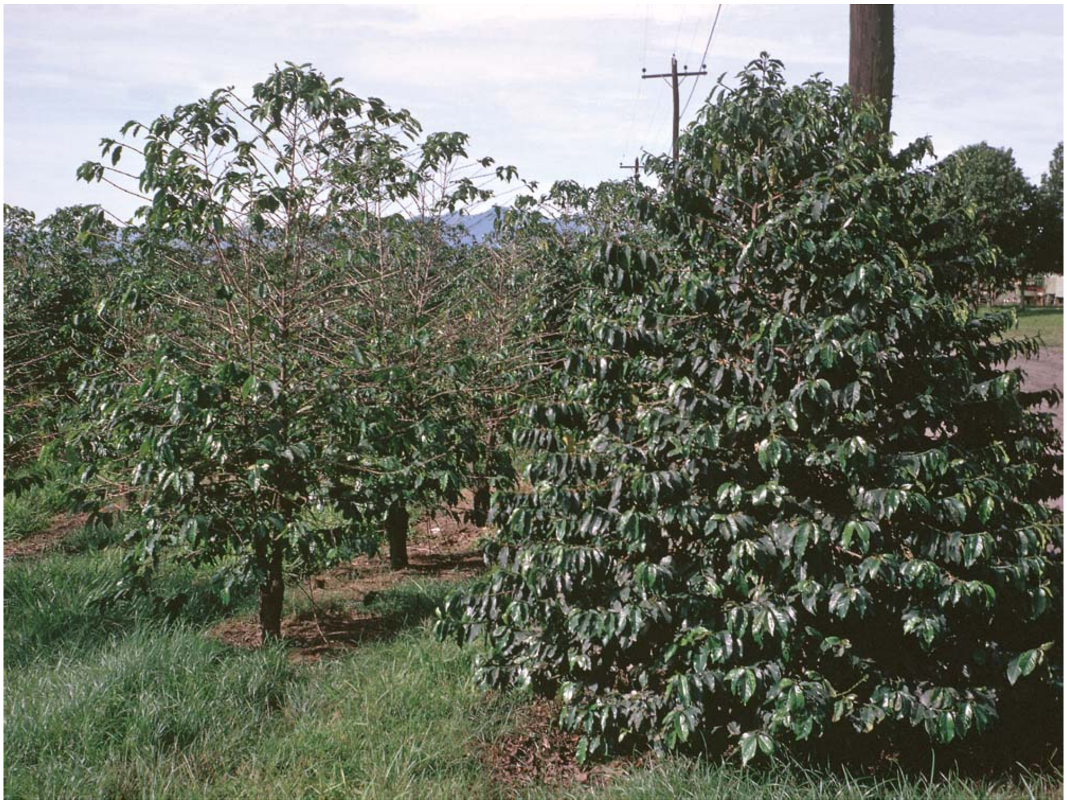


Figure 10.3. *Coffea arabica* (left) defoliated by coffee rust, a polycyclic disease, compared with a resistant hybrid of *C. arabica* and *C. canephora* (right).

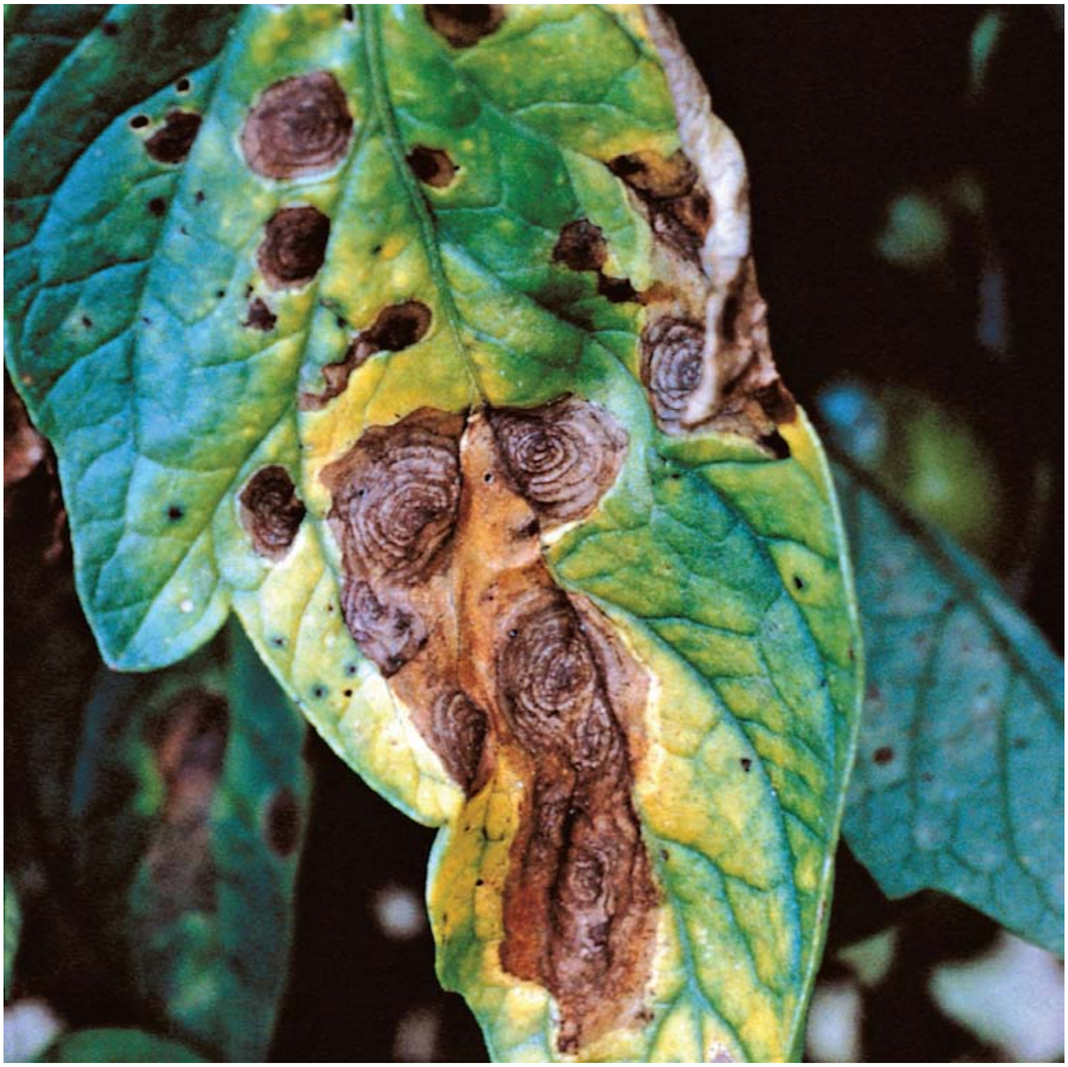


Figure 10.4. Early blight (*Alternaria solani*) of potato and tomato, a polycyclic, foliar fungal disease. Note the concentric rings in the lesions, characteristic of *Alternaria* diseases.

Epidemiology is the study of disease increase in populations. There are two basic models for plant disease epidemiology: one for monocyclic pathogens and another for polycyclic pathogens. Monocyclic pathogens complete only one disease cycle during a growing season and can be managed by

limiting primary inoculum. Polycyclic pathogens complete several to many disease cycles during a growing season. It may be necessary to manage both primary inoculum and the rate of infection caused by a polycyclic pathogen.

The Disease Progress Curve

The **disease progress curve** tracks the amount of disease in an epidemic over time (Figure 10.5). Time is plotted on the *x*-axis, and some measure of disease is plotted on the *y*-axis. Various parameters may be used to represent a measure of disease: the number of plants infected, the proportion or percent of plants infected, the percent of leaf area showing symptoms, the percent of defoliation, or the amount of inoculum produced.

How high will the line go on a disease progress curve for a monocyclic epidemic? The important determinants are the amount of primary inoculum and how much susceptible tissue or how many healthy plants remain to be infected. For example, if nematode eggs or microsclerotia of *Verticillium* are abundant in the soil, the proportion of infected plants will be higher than in soils with less inoculum, and the line might reach 100% disease. If an attempt is made to reduce primary inoculum or its efficacy in causing infection, the level of disease will depend on how successful the management practice was.

In polycyclic epidemics, a few to multiple generations of inoculum are produced in a single growing season. Primary inoculum begins the epidemic, but secondary inoculum also is produced. Reducing the amount or efficacy of the primary inoculum is important, but management practices that focus on reducing the rate of infection also are usually required.

DISEASE CLASSIC

Coffee Rust

Hosts Cultivated coffee trees (*Coffea arabica* and *C. canephora* var. *robusta*) and wild *Coffea* species (telial hosts). No aecial host is known.

Pathogen *Hemileia vastatrix*, a rust fungus (basidiomycete) that is apparently heteroecious, although only teliospores, basidiospores, and urediniospores have been observed on coffee.

Symptoms and Signs Pale yellow spots form on leaves. Later, uredinia filled with powdery orange urediniospores are produced through stomata on the lower surface of the leaf. Heavily infected leaves drop prematurely. Teliospores form later where urediniospores were produced.

Disease Cycle Teliospores and basidiospores are produced from the same mycelium that produces urediniospores but play no known role in the disease cycle. Urediniospores serve as inoculum. They are dispersed through the air from infected leaves, including leaves that have fallen prematurely as a result of the disease. Free water is required for infection, and the spores infect only through stomata. *H. vastatrix* is a polycyclic pathogen of a tropical tree that retains its leaves throughout the year.

Management Fungicides are used to protect trees from rust in most commercial production areas. It is difficult to find durable genetic resistance because the pathogen is polycyclic, and the tree hosts are long-lived. Some cultural practices can reduce disease, but they vary depending on the location of the trees.

Significance Coffee and the coffee rust fungus probably originated in Ethiopia. The early coffee plantations planted by European colonists were in Southeast Asia, including the large island of Java (now part of Indonesia), and the name *java* came to be used as a slang term for coffee. The coffee rust epidemic in Ceylon (Sri Lanka) led to tea becoming the hot beverage of choice for the British. To escape the disease, most of the world's coffee is now grown in the Western Hemisphere. However, coffee rust became established there in the 1970s.

Plant Disease Profiles: Coffee rust

The rate of infection depends on how many generations of secondary inoculum are produced and its efficacy in initiating new infections. The epidemic will continue as long as both inoculum and susceptible tissue are available, perhaps for the entire growing season. The line of a disease progress curve could potentially reach 100% disease (Figure 10.5A). Although many agricultural areas have growing seasons that end in winter or a dry season, many areas in the tropics have year-round crop production during which diseases may be active. Polycyclic pathogens can be particularly devastating in these areas where epidemics can develop over more than a single season or year.

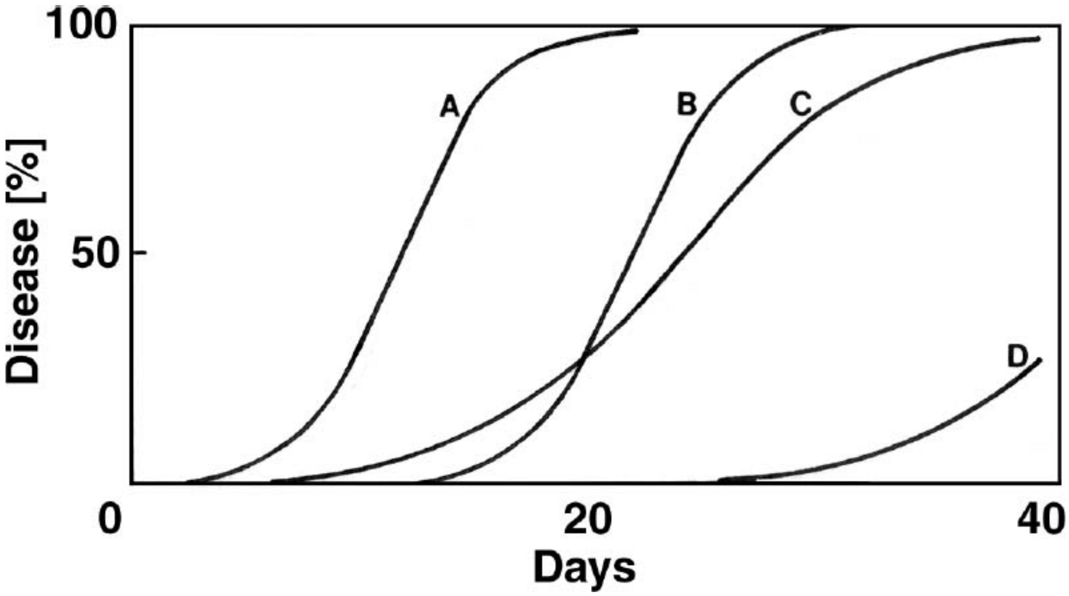


Figure 10.5. Disease progress curves of a polycyclic pathogen under various conditions. A = no management. B = reduction of primary inoculum, which delays the epidemic but does not reduce the rate of infection. C = reduction in the rate of infection. D = combination of B and C, i.e., the epidemic is delayed and the rate of infection is reduced.

The Mathematics of a Polycyclic Epidemic

The relative importance of initial or primary inoculum and rate of infection can be seen from a mathematical perspective in the solution to the differential equation that describes the increase of disease in polycyclic epidemics:

$$x = x_0 e^{rt}$$

where

x = the proportion of disease

x_0 = the proportion of disease at the beginning of the season (sometimes loosely described as the amount of initial, or primary, inoculum)

r = rate of infection

t = time

The amount of disease increases linearly with the amount of initial disease, but the rate of development increases exponentially with time.

Disease progress curves represent plant disease epidemics by plotting disease vs. time. In both monocyclic and polycyclic epidemics, disease may reach 100%, but the final outcome is related to many factors affecting the plant, the pathogen, and the environment.

Effects of Management Practices on Disease Progress Curves of Polycyclic Epidemics

Polycyclic pathogens are managed by reducing the amount or efficacy of primary inoculum and/or by reducing the rate of infection. A banking analogy can explain how management practices affect the disease progress curve. If you and a friend both establish savings accounts, but you start your account with twice as much money as your friend, you will have more money at the end of the year even though both accounts earn the same rate of interest.

Management practices that reduce primary inoculum are like starting a savings account with less money. The disease progress curve shifts to the right in proportion to how much the inoculum or its efficacy was reduced (Figure 10.5B), but it does not affect how quickly the line rises, i.e., the slope of the line. Just as the rate of interest is the same no matter how much money is used to establish a savings account, reducing inoculum does not change the rate of infection. The epidemic is delayed, but a severe epidemic may still occur if environmental conditions are favorable.

Management practices that reduce the rate of infection reduce the ability of inoculum to cause infection throughout the time the plant is susceptible. Examples include applications of a fungicide, minimizing leaf wetness, or using a cultivar with general resistance. Less disease occurs with each generation of secondary inoculum, and/or the number of generations is reduced. Because the rate of infection is reduced, the slope of the disease progress curve is reduced (Figure 10.5C). Using the banking analogy again, this is like starting two savings accounts with the same amount of money (the same primary inoculum), but with a higher interest rate for one account than for the other. The account with the higher interest rate will increase more quickly. When environmental conditions are optimal for a pathogen, both primary inoculum and secondary inoculum will have more opportunities to initiate infections. There may be more generations of secondary inoculum, and the rate of infection increases. The curve climbs quickly and levels off only when no more susceptible tissue is available or all of the plants are dead.

Integrated disease management uses a combination of practices to reduce both primary inoculum and the rate of infection. Less primary inoculum delays the epidemic and shifts the disease progress curve to the right. A reduced rate of infection slows the epidemic and reduces the slope of the disease progress curve. When combined, disease is minimized (Figure 10.5D). This is why integrated management programs are the most effective.

Management practices that reduce the amount or efficacy of primary inoculum of a polycyclic pathogen shift the disease progress curve to the right (delaying the epidemic), but they have no effect on the rate of infection. Management practices that reduce the rate of infection also reduce the slope of the curve. Combinations of practices that focus on reducing both primary inoculum and the rate of infection (integrated disease management) are most effective.

Comparing Epidemics

It is often useful to compare epidemics to optimize management practices. For example, the use of a biological control agent may be compared to a chemical fumigant for reducing primary inoculum. General (horizontal) resistance may be compared to the use of fungicide to reduce the rate of infection. One way to compare epidemics is to compare their rates of development. Mathematics can be used to calculate an apparent infection rate from disease progress graphs, and these rates can be compared statistically.

Another common means of comparing epidemics is to measure the “area under the disease progress curve” (AUDPC) (Figure 10.6). This measure of the total amount of disease during the epidemic can be calculated by crude estimates, such as counting the squares on graph paper under the curve, or by using computer programs that calculate the AUDPC. The AUDPC also can be used for statistical analysis of epidemic comparisons.

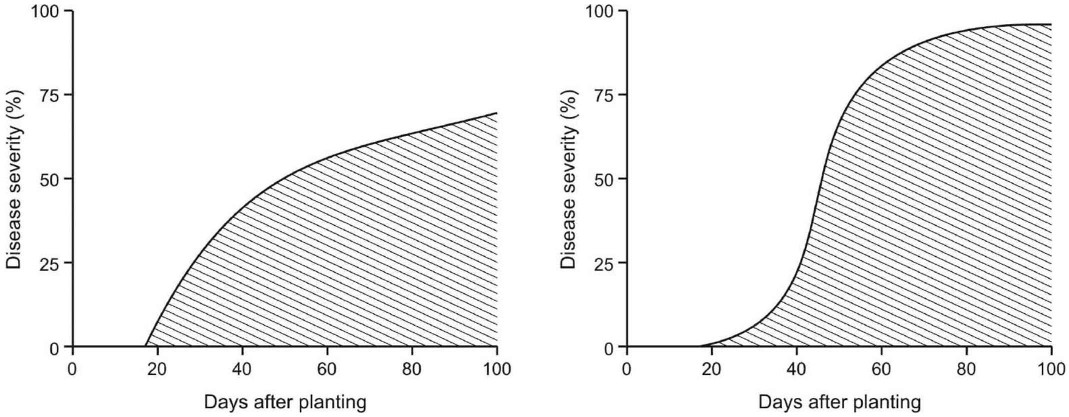


Figure 10.6. Epidemics can be compared by calculating the area under the disease progress curve (AUDPC, shaded area) for each epidemic. These are two examples.

The most common way to compare polycyclic epidemics is to calculate the area under the disease progress curve (AUDPC).

This permits plant pathologists to evaluate the effectiveness of various management practices.

Progress of Polycyclic Epidemics

Theoretically, any polycyclic epidemic will continue until all of the plants are dead, but there is not always enough time for this to happen. Several factors determine the rate of infection, i.e., how quickly the curve reaches its maximum (potentially 100%): the number of generations of secondary inoculum, the susceptibility of the plants, and the environmental conditions.

Pathogens can produce enormous amounts of primary and secondary inoculum. For example, *Phytophthora infestans* may produce 100,000 sporangia from a single foliar lesion on a potato plant. A single uredinium of *Puccinia graminis* on a wheat leaf may produce 2,000 urediniospores per day for two to three weeks. When a strong weather front approaches a wheat farm, nearly 4 million viruliferous aphids may arrive carrying *Barley yellow dwarf virus*. Despite these large numbers, most of the inoculum will not be successful in establishing new infections. For fungi and bacteria in particular, temperature and moisture determine how quickly each generation of secondary inoculum is produced and how successful the inoculum will be in producing new infections.

Notice that the time axis of the disease progress curve (Figure 10.5) does not indicate the point during a growing season when the epidemic begins. Some epidemics begin early in the growing season, but plant tissue is not susceptible to infection for the entire time. For example, only young, expanding apple leaf tissue is susceptible to infection by the apple scab pathogen; mature leaf tissue is not. In other cases, disease does not start until later in the season, such as when the leaf canopies of mature potato plants close and enhance the moist conditions necessary for late blight. Plants may become more or less susceptible depending on nitrogen fertilizer applications. Environmental conditions may enhance or reduce the opportunity for production and dispersal of secondary inoculum and its ability to initiate new infections.

How far and how quickly a polycyclic epidemic progresses in a season is governed by characteristics of the plant, the pathogen, and the environment.

Reducing Primary Inoculum or Reducing Rate of Infection—Relative Importance for Polycyclic Pathogens

Apple scab, citrus canker (Figure 10.7), coffee rust, late blight of potato and tomato, and stem rust of wheat are often used as examples of polycyclic epidemics because of their tremendous potential to cause disease. If the rate of infection is very high, then reducing primary inoculum has only a small impact on reducing disease.

DISEASE CLASSIC

Citrus Canker

Hosts *Citrus* species, cultivars, and hybrids, including orange, grapefruit, lemon, and lime

Pathogen *Xanthomonas axonopodis* pv. *citri* and *X. axonopodis* pv. *aurantifolii*

Symptoms and Signs The bacteria infect mostly leaves and fruits but sometimes twigs. Leaf lesions usually have a bright yellow halo, and they become corky and sunken on the lower leaf surfaces. As lesions accumulate, leaves may fall. Fruit lesions are similar to leaf lesions. Stem lesions become corky and may be a survival site for the bacteria.

Disease Cycle The bacteria infect through stomata and wounds. Splashing water distributes the bacteria to new infection sites. Wind-blown rain can carry the bacteria.

Management In the United States, exclusion and eradication were the primary methods of control. After the reappearance of the disease in Florida in 1984, millions of dollars were spent for inspections and destruction of infected trees and all other susceptible trees within a 580-meter (1,900-foot) radius of an infected tree. Hurricanes that occurred in 2004 and 2005, however, spread the bacteria across the entire state, and the eradication program was ended. Where the disease is endemic, only more-resistant types of citrus can be grown. Cultural practices include the use of windbreaks to reduce spread of the bacteria and using care in pruning and other practices that create wounds. Copper-containing sprays are used to protect trees when they are most susceptible to infection. This is a difficult disease to manage because it is a polycyclic pathogen on a long-lived tree growing in a warm, wet climate that favors pathogen development.

Significance Citrus canker probably originated in Southeast Asia, the center of origin of *Citrus*. The disease is endemic in most citrus-growing areas of the world. In the United States, it is a problem only in Florida.

Plant Disease Profiles: Citrus canker

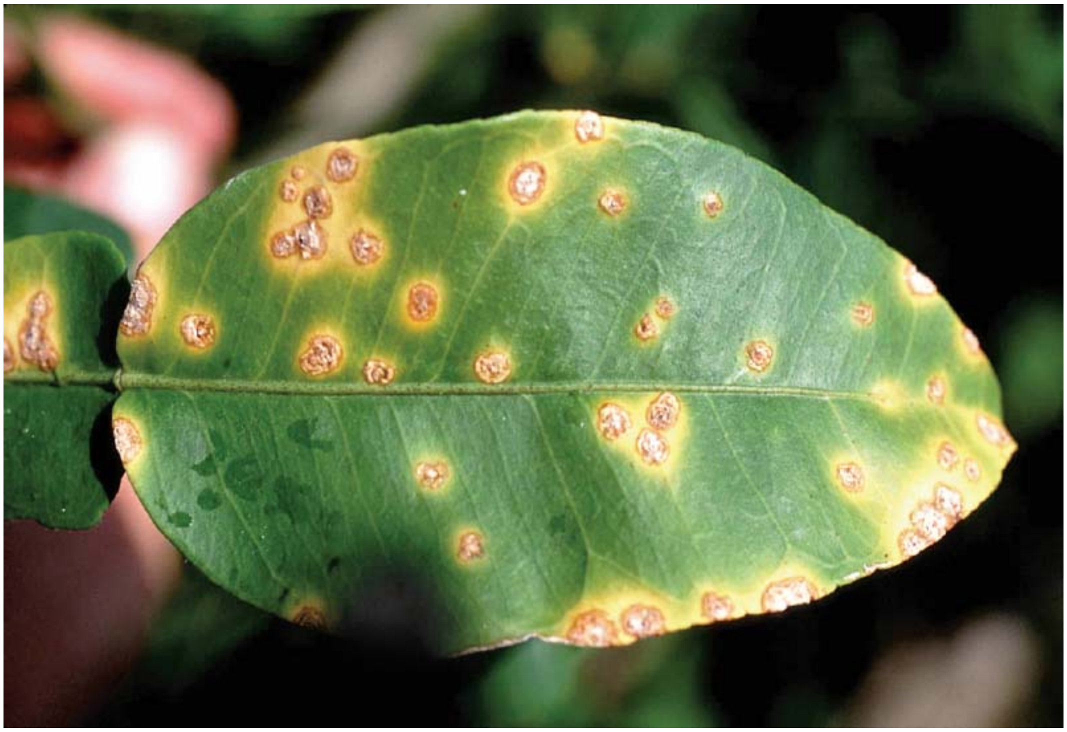


Figure 10.7. Foliar symptoms of citrus canker, a polycyclic disease. Note the halo around each lesion, typical of a bacterial disease.

However, several polycyclic pathogens have relatively low rates of infection. Many so-called “minor diseases” are caused by polycyclic pathogens. Also, some polycyclic pathogens are active only during short periods of time when primary inoculum is produced or a plant is susceptible. Other polycyclic pathogens are in environments, such as dry areas, in which opportunities for infection are limited.

If secondary infection cycles develop slowly (i.e., the rate of infection is lower), then reducing primary inoculum becomes more important. Shifting the disease progress curve to the right may be sufficient to keep the final amounts of disease at the time of harvest to an acceptable level. Also, if there are suitable rate-reducing management alternatives, such as modification of environmental conditions or partial genetic resistance, then reducing primary inoculum may have a bigger impact on the final amount of disease.

Reduction of the amount or efficacy of primary inoculum is most successful for polycyclic epidemics with low rates of infection. If the rate of infection is high, rate-reducing activities throughout the growing season may be necessary. Epidemics progress only when the pathogen is active and plants are susceptible. The final impact of an epidemic is determined by the time of harvest or when the growing season ends.

Can we predict plant diseases?

With insect pests, it is often recommended to wait until the population reaches a predetermined economic damage threshold before applying a management measure, such as an insecticide spray. With plant diseases, we usually cannot detect the primary inoculum and must wait until visible symptoms develop. By the time symptoms are observed, it is often too late to effectively stop a disease epidemic. Researchers have tried to create predictions, also called forecasts or warnings, to help determine whether and when preventive management measures are needed.

Kinds of Diseases that Can Be Predicted

Most forecast systems have been created for high value crops for which we have effective disease-management strategies. The disease should be economically important, but sporadic—worse in some years than others. If disease pressure is uniform each year, prediction is less valuable. It also is important that growers or plant managers be able to operate the prediction system themselves, or that there is a good means of communication between those who monitor the disease and those who need to take action to manage it.

Characteristics of Plant Diseases That Are Good Candidates for Prediction

- economically important
- sporadic
- disease management strategies available
- growers can monitor predictions themselves or receive timely notification

Prediction of Monocyclic Epidemics

The severity of a monocyclic epidemic is related to the amount or efficacy of the primary inoculum. Forecasting systems for monocyclic pathogens are usually based on some measure of primary inoculum. Sometimes this can be accomplished by a simple bioassay. For example, Aphanomyces root rot of peas is sometimes a problem when there is a large population of oospores in the soil from the previous season. Peas can be planted in soil samples in a greenhouse during the winter months to determine whether the population of the pathogen is high enough to be economically important in the coming season. Soils may be assayed for soybean cyst nematode eggs and/or cysts to determine whether a resistant cultivar or a nonhost crop should be planted (see Figures 4.14 and 4.15). Other pathogen survival structures have been assayed in soils, including the sclerotia of *Sclerotinia sclerotiorum* (white mold) and *Sclerotium rolfsii* (southern blight) and microsclerotia of *Verticillium dahliae*.

Predictions of Monocyclic Diseases

- predict amount or efficacy of **primary inoculum**
- **examples:** nematode eggs in soil, fungal sclerotia in soil

Prediction and Management of Soybean Cyst Nematode

If population density (number of eggs per 100 cubic centimeters of soil) of the soybean cyst nematode (SCN) is high, plant a nonhost crop until the population density is below the threshold (the threshold varies with local conditions).

If the SCN population density is below the threshold, a six-year rotation may be followed:

- year 1: nonhost crop
- year 2: resistant soybean cultivar
- year 3: nonhost crop
- year 4: soybean cultivar with different resistance gene(s) than used in year 2
- year 5: nonhost crop
- year 6: susceptible cultivar

Even though many monocyclic pathogens are soilborne, some are foliar pathogens. Cedar-apple rust (*Gymnosporangium juniperi-virginianae*) infections occur only when temperature and moisture conditions favor production and release of basidiospores from galls on juniper trees (Figure 10.8). These factors also determine when young apple or crabapple leaves are susceptible. Environmental factors can be monitored to optimize the application of fungicides to apple or crabapples leaves. In warm, dry springs, infection is likely to be low, and fungicides may not be needed at all.

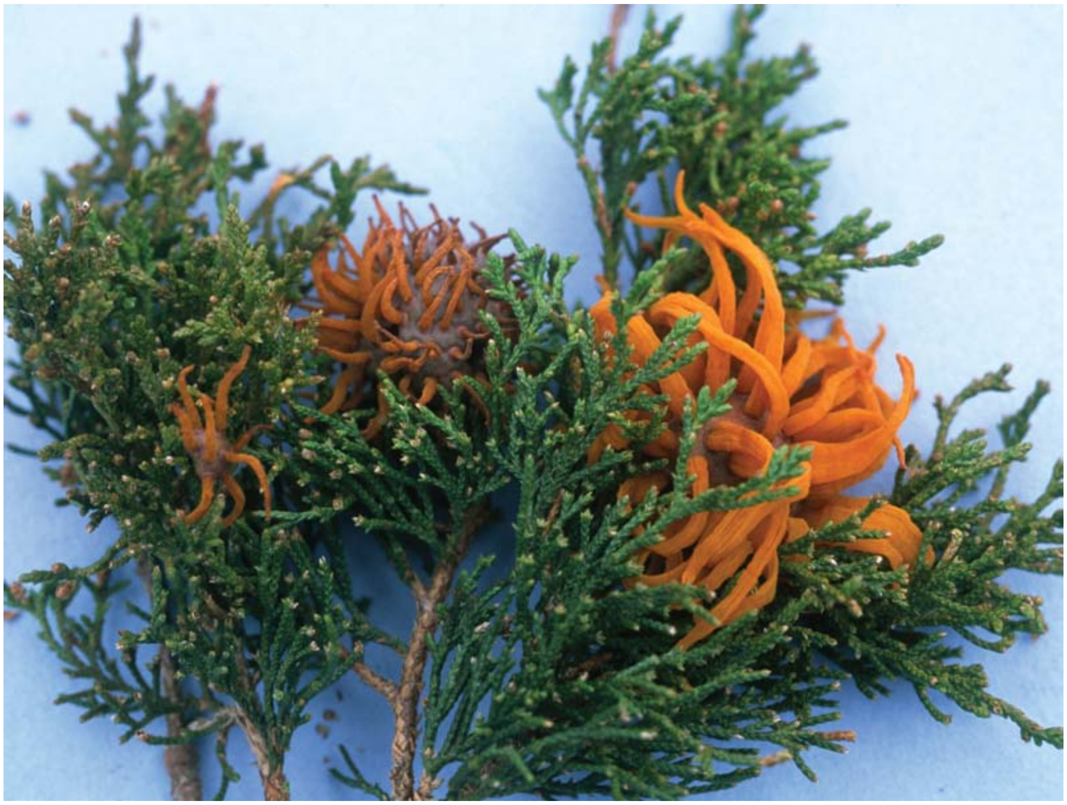


Figure 10.8. **The production of basidiospores from telial galls of the cedar-apple rust fungus on juniper (red-cedar) is used to determine the need for and the timing of application of protective fungicides to apple trees.**

Prediction of Polycyclic Epidemics

Some economically important diseases, such as apple scab, late blight of potato and tomato, and peanut leaf spots, require the use of fungicides since the potential for disease development continues over an extended period of time. Therefore, growers need to know every time that environmental conditions favor infection. The time required for infection to occur, usually hours of leaf wetness at a particular temperature, is called an **infection period**.

Infection period: the time required for infection to occur, usually hours of leaf wetness at a particular temperature

In 1944, W. D. Mills first published a table of infection periods for apple scab in which he noted how many hours of leaf wetness were required for germination of ascospores at the optimal temperature. Infection periods require longer periods of leaf wetness when temperatures are above or below the optimum. If growers receive “ascospore reports” of the initiation, peak, and end of ascospore release, they can then monitor the environment during the ascospore release period and protect trees with fungicides when infection periods occur. This system predicts the efficacy of primary inoculum (ascospores). In years with few infection periods, fewer fungicide applications are needed. If trees have been protected from scab until the end of ascospore release (usually about the end of flowering), additional fungicides are not needed to protect leaves and fruit against secondary inoculum for the rest of the growing season.

Predictions or Warnings for Most Polycyclic Diseases

- predict infection periods for secondary cycles (whenever plants are susceptible)
- calculate severity values based on accumulating infection periods

The “Mills Table” for Apple Scab Infection as Modified by A. L. Jones, Michigan State University

Modified and reprinted from Plant Disease Profiles: Apple scab

Wetting Period Required for Apple Scab Infection at Different Air Temperatures, and Time Required for Development of Conidia by Lesions at Different Air Temperatures

Average temperature		Wetting period (hours) for heavy infection	Incubation period (days)
(°F)	(°C)		
63–75	17.2–23.9	18	9
62	16.7	19	10
61	16.1	20	10
60	15.6	20	11
59	15.0	21	12
58	14.4	21	12
57	13.9	22	13
56	13.3	22	13
55	12.8	24	14
54	12.2	24	14
53	11.7	25	15
52	11.1	26	15
51	10.6	27	16
50	10.0	29	16
49	9.4	30	17

Similar infection period prediction systems have been developed for many foliar polycyclic pathogens that are typically managed with fungicides, including those that cause anthracnoses, downy mildews, leaf spots, and rusts. For many of these diseases, we assume that primary inoculum is present each year and monitor only the environmental conditions that favor infection.

Stewart’s wilt of corn (*Erwinia stewartii* syn. *Pantoea stewartii*) is a bacterial disease in which the pathogen is disseminated by corn flea beetles. The bacteria survive winter in association with the beetles, so beetle survival can determine whether a springtime insecticide application to sweet corn is warranted. For this polycyclic pathogen, the environmental conditions that affect survival of the vector are monitored. N. E. Stevens first correlated winter temperatures with flea beetle survival and disease risk in the 1930s. Fifteen years later, G. H. Boewe refined Stevens’ work, so growers would know whether economically important levels of infection were likely to occur in susceptible corn cultivars. The growers could then choose to apply an insecticide at the time of vector emergence. This was one of the earliest practical disease-prediction systems.

For some diseases, prediction systems warn growers each time that an infection period occurs or is anticipated. For other diseases, infection periods accumulate to create **severity values**. A designated severity value can be used as an action threshold for a fungicide application or another disease management activity. An advantage of a severity value system is that the action threshold can be modified by the grower according to local conditions and/or disease tolerance.

Both the production of secondary inoculum and the number of generations of secondary infections are determined by environmental conditions, so computer models have been created that permit growers to monitor the environment and receive warnings. Some growers use computerized weather stations that predict certain diseases based on their local conditions (Figure 10.9). Commercial companies are now using global-positioning systems (GPS) to provide growers with precise environmental reports and sometimes with specific disease predictions.

Examples of Diseases for Which Prediction Systems Have Been Developed

- anthracnose, dollar spot, brown patch, and Pythium blight of turfgrass
- apple scab
- blue mold (downy mildew) of tobacco
- Botrytis leaf blight of onion (Botcast)
- Cercospora on celery
- early blight of tomato (FAST—Forecasting *Alternaria solani* on tomato)
- late blight of potato (Blitecast)
- leaf spots on peanut
- Septoria tritici leaf blotch of wheat

Some of these prediction models have been combined into crop-management programs that include several diseases and insect pests:

- EPIPRE—winter wheat in Europe
- WISDOM—potato management in Wisconsin

Uses of Disease Prediction

- to notify growers when an infection period occurs
- to improve efficacy of fungicides, other disease management chemicals, and biological control agents (apply exactly when infection is likely to occur)
- to reduce pesticide use compared to schedules set by the calendar (in years when less is needed)
- to establish disease thresholds or severity values for the practice of integrated disease management
- to verify scientifically the need for pesticide application, to satisfy government regulators (without waiting for symptom

development)

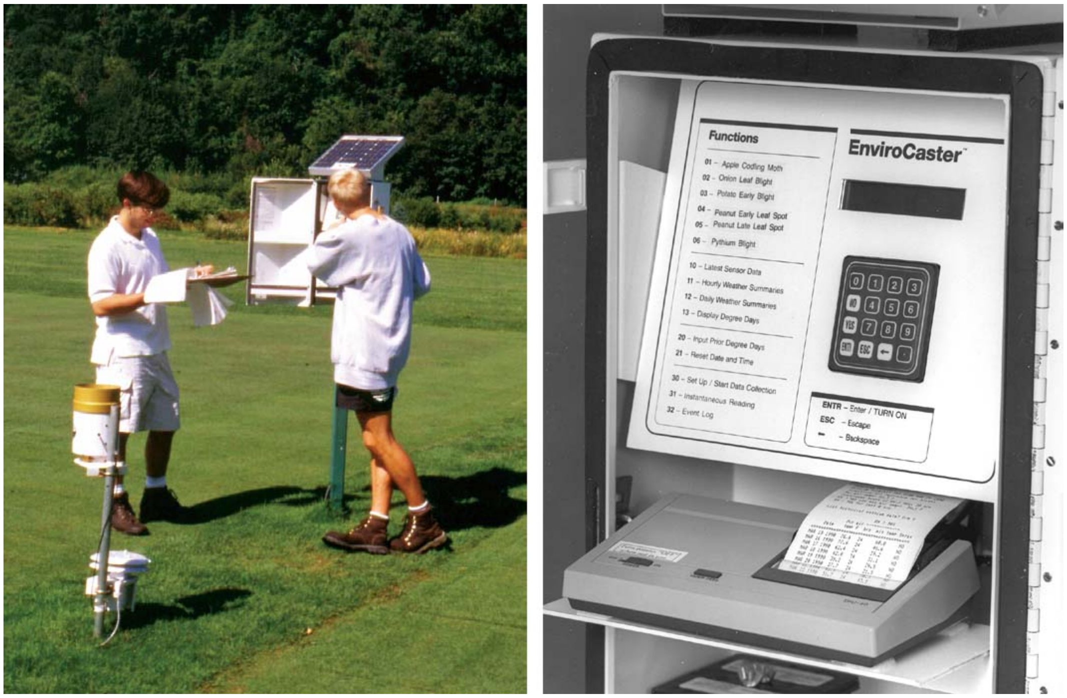

Functions
01 – Apple Codling Moth
02 – Onion Leaf Blight
03 – Potato Early Blight
04 – Peanut Early Leaf Spot
05 – Peanut Late Leaf Spot
06 – Pythium Blight
10 – Latest Sensor Data
11 – Hourly Weather Summaries
12 – Daily Weather Summaries
13 – Display Degree Days
20 – Input Prior Degree Days
21 – Reset Date and Time
30 – Set Up / Start Data Collection
31 – Instantaneous Reading
32 – Event Log
EnviroCaster
ENTR – Enter / TURN ON
ESC – Escape
← – Backspace

Figure 10.9. Computerized environmental-monitoring equipment can be used with models that predict disease outbreaks, based on environmental data for turfgrass and various other crops.

Disease prediction is most useful for economically important, sporadic diseases for which we have effective management strategies and a means to communicate with growers. Monocyclic epidemics are predicted by monitoring the amount or efficacy of primary inoculum. Predictions of polycyclic epidemics are usually based on infection periods governed by leaf moisture and temperature. Environmental factors that affect vector survival are sometimes used in disease predictions. Infection period predictions may be used to calculate severity values, which can then be used to establish action thresholds for disease management.

How do people affect the susceptibility of plants to disease?

Multiple plant species are usually present in natural ecosystems (Figure 10.10A). If a pathogen produces inoculum, its nearest neighbors may not be related plants and susceptible to infection. Even plants of the same species are likely to be genetically variable due to genetic recombination. Some of these plants might be susceptible to new infections, but others might have some level of resistance. Although storms and animals may cause wounds, natural ecosystems are not generally exposed to routine cutting, pruning, harvesting, tilling of soil, weeding, or other agricultural activities that create wounds and distribute inoculum.

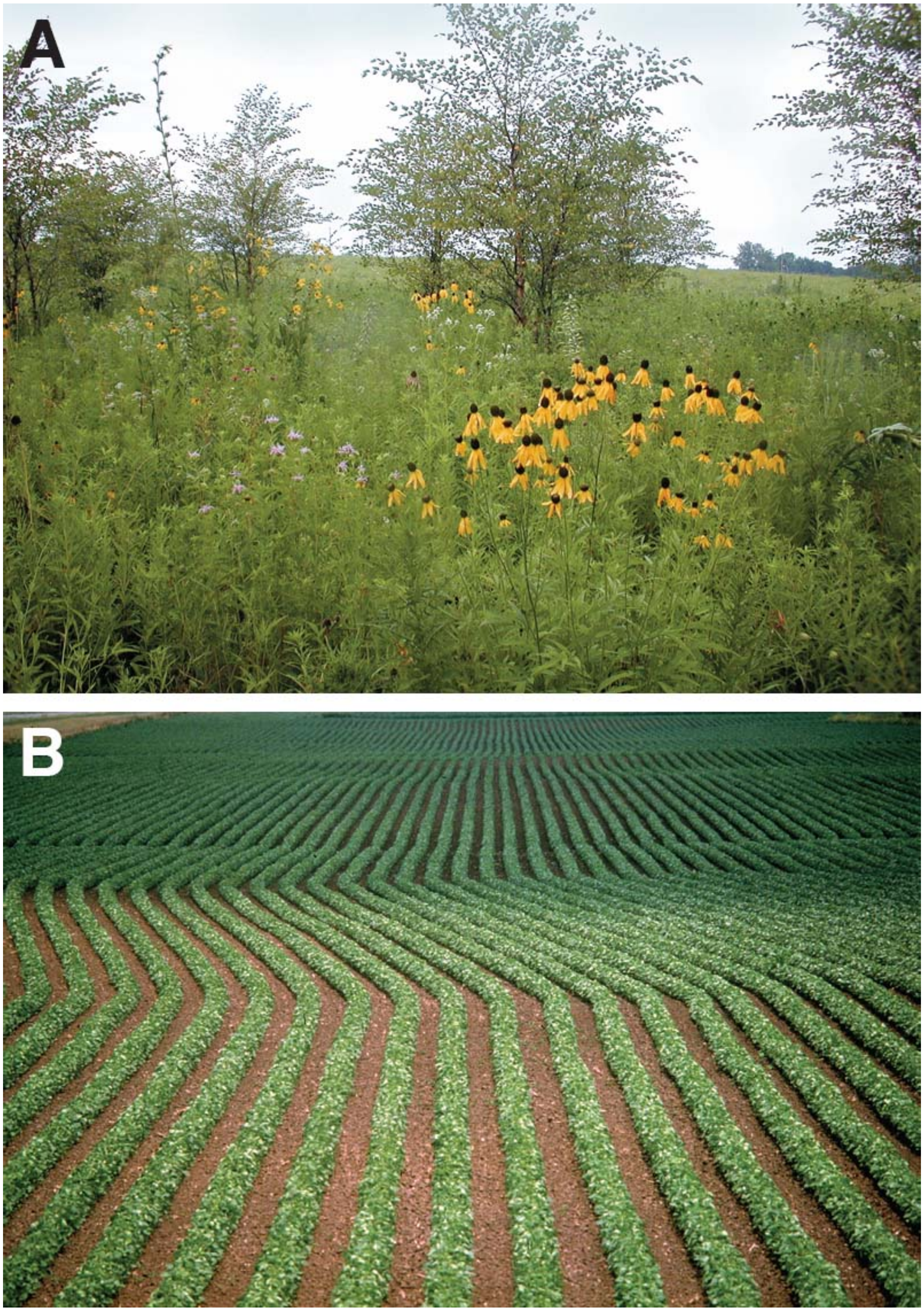
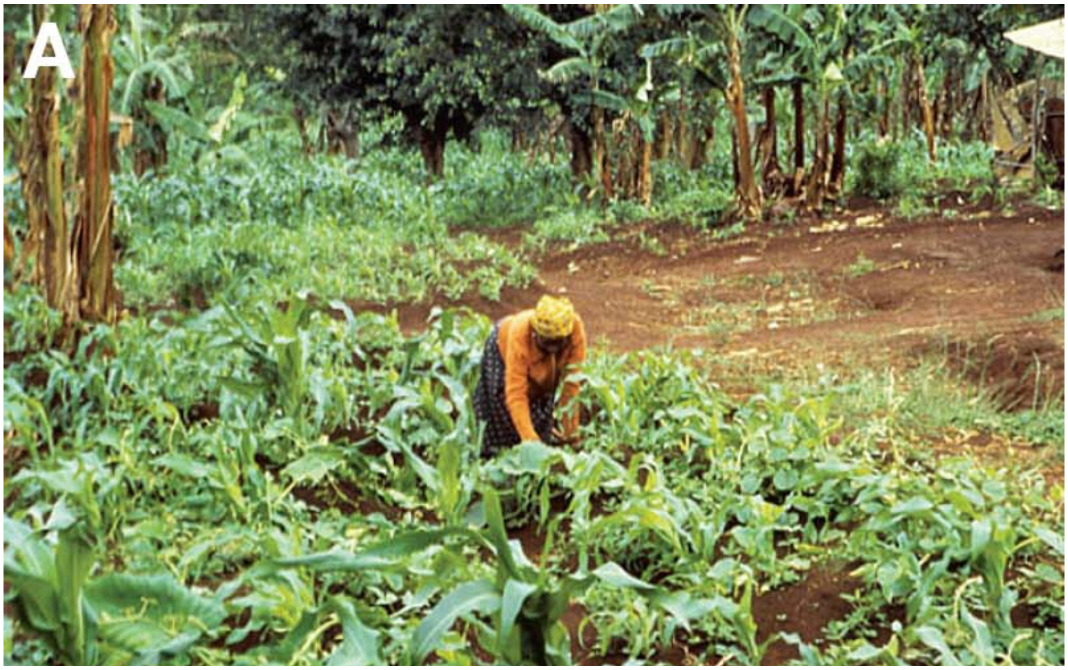


Figure 10.10. Ecosystems of the U.S. Midwest. A, Natural ecosystem: a tall-grass prairie. B, Agro-ecosystem: a soybean field.

Native pathogens in natural ecosystems generally live in their **center of origin**. The native plants have lived in association with the pathogens for long periods of time and have developed some level of resistance to them. These localized pathogens are not a threat to potentially susceptible plants across mountain ranges or across bodies of water on other continents. The following factors cause agroecosystems to be different from natural ecosystems and increase the potential of pathogens to cause epidemics.

Monoculture

In agriculture, we typically see large expanses of land planted to a single species (Figures 10.10B and 10.11B) instead of diverse plant communities (Figure 10.11A). The spacing between plants of the same species is greatly reduced because this simplifies planting, cultivation, and harvesting practices, thereby reducing productions costs. If a pathogen infects one of these plants, the inoculum it produces, whether airborne, waterborne, or vectorborne, has a much greater probability of coming into contact with a suitable host than inoculum produced on plants in diverse natural ecosystems.



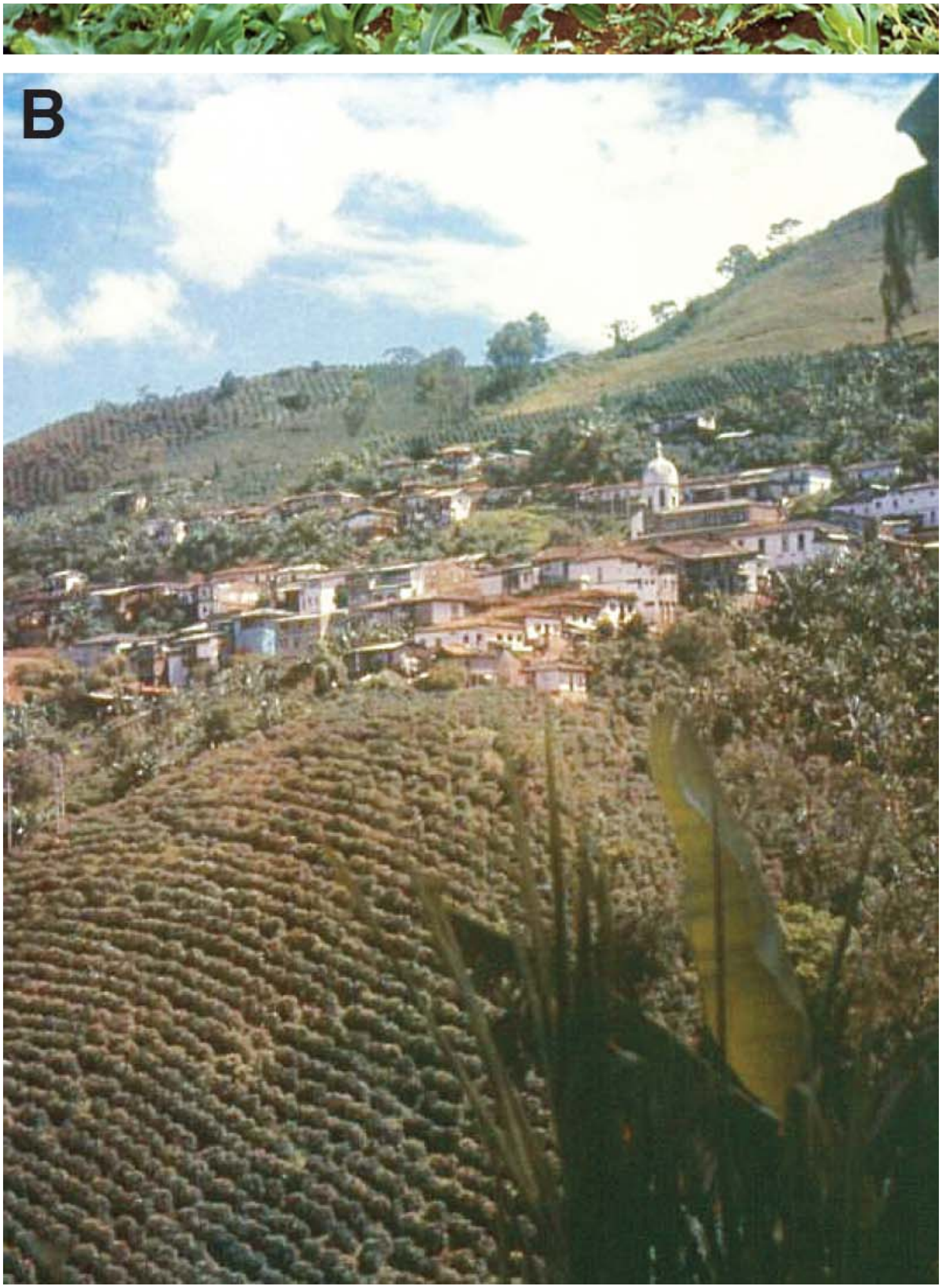


Figure 10.11. A, Polyculture: mixed cropping (seven different crops) in Africa. B, Monoculture: a coffee plantation in Colombia.

Both the coffee plant and the coffee rust pathogen (*Hemileia vastatrix*) are believed to have originated on the African continent, probably in southern Ethiopia. In the wild, the widely spaced coffee trees are relatively unaffected by the pathogen. However, when people began to grow coffee in monocultures on plantations in Southeast Asia and later in South America, devastating epidemics of rust developed, destroying many hectares of closely spaced trees. Urediniospores were easily and rapidly blown by air currents to neighboring trees in the plantation monocultures, initiating many secondary infection cycles that resulted in defoliation and eventual death of the trees.

► **DID YOU KNOW?**

The British consume approximately half of the tea produced in the world. They became tea drinkers when plantation owners in their colony of Ceylon (now Sri Lanka) were forced to abandon formerly lucrative coffee plantations that were devastated by coffee rust in the 1870s. In 1875 there were 160,000 hectares (400,000 acres) of coffee but only 400 hectares (1,000 acres) of tea on the island. Within 5 years, as rust killed the coffee trees, tea had been planted on 120,000 hectares (300,000 acres), providing plenty of this beverage for thirsty and patriotic British citizens.

For certain crops, monocultures can extend across continents. Large expanses planted to corn, potatoes, rice, soybeans, and certain timber species are vulnerable to infection by pathogens carried easily from planting to planting by wind or vectors without respect for political borders or land ownership.

Introduced Plants

People have moved many plant species from their centers of origin to distant parts of the world (Figure 10.12). If we had access only to plants that were native to where we live, our diets would be much less interesting and many areas of the world would not be inhabited. In addition, exotic flowers, turfgrasses, and woody ornamentals add beauty and color to our environments.

► **DID YOU KNOW?**

Nikolay Vavilov (1887–1943), a Russian geneticist, proposed that areas where wild relatives of plant species occur are most likely the sites of their original domestication. These areas are also their centers of biological diversity and are important sources of new genes for crop improvement, including disease resistance. Vavilov identified eight major centers of crop domestication.

China
apple, onion, peach, soybean

India
banana, pepper, rice, sugar cane, and banana in the Indo-Malayan center

Central Asia
carrot, cotton, grape, pea, mustard, wheat

Near East
alfalfa, cherry, oat, rye, wheat

Mediterranean
beet, lettuce, spice plants, wheat

Abyssinia
coffee, sorghum, wheat

South Mexico and Central America

amaranth, cherry tomato, cotton, maize, pepper, sweet potato

South America
cashew, lima bean, peanut, pepper, pineapple, potato, tobacco, tomato

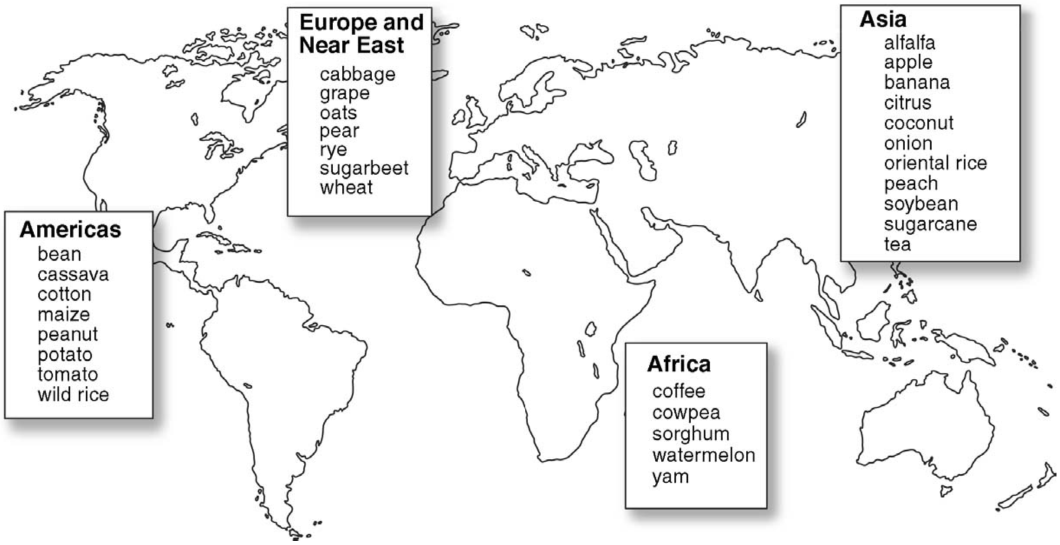

Europe and Near East
cabbage
grape
oats
pear
rye
sugarbeet
wheat
Asia
alfalfa
apple
banana
citrus
coconut
onion
oriental rice
peach
soybean
sugarcane
tea
Americas
bean
cassava
cotton
maize
peanut
potato
tomato
wild rice
Africa
coffee
cowpea
sorghum
watermelon
yam

Figure 10.12. A map of sites of early plant domestication reveals how people have moved many important crops around the world.

People have probably always carried plants and seeds to new living sites, but most of the early migrations were relatively short. The voyages of Columbus and other explorers initiated a slow, long-distance biological exchange between the Western and Eastern Hemispheres. Many plants and seeds carrying their pathogens and insect pests rotted or were eaten before the ships reached port, but eventually many new crops were introduced to both hemispheres. As human travel and interest in new plants for trade and food supplies increased, the movement of plant materials around the world also increased. In the early 1800s, U.S. President John Quincy Adams explicitly encouraged people to import potentially useful plants; laws restricting plant movement into the United States were not enacted until the early 1900s (see Chapter 11).

> “The President is desirous of causing to be introduced into the United States all such trees and plants from other countries not heretofore known in the United States as may give promise under proper cultivation, of flourishing and becoming useful.”
>
> —John Quincy Adams, 1827

With the increased movement of plants around the world, it is not surprising that sometimes a plant was introduced into an area where it was susceptible to a native pathogen. *Erwinia amylovora* is a native North American bacterium that causes a minor disease on native rosaceous plants such as brambles, hawthorn, mountain ash, and rose. When Europeans introduced apple and pear trees to North America, these plants had no natural resistance to the bacterium and quickly succumbed to a disease so severe that it was named fire blight. Pears are particularly susceptible and today are primarily produced in the dry, western environments of Washington and Oregon, where the disease is more easily managed. Fire blight remains a serious disease of the genetically uniform apple orchards found throughout North America. More recently, *E. amylovora* was accidentally introduced into Great Britain (1957) and Europe (1966), and it continues to spread to apple and pear orchards around the world.

Genetic Uniformity

Plants in agricultural settings are likely to be grown from seed or propagative parts from plants that have been selected over generations to have very similar genetic traits. These are desirable in agriculture. When wheat plants in a field all mature at the same time and height, they can be mechanically harvested. People often prefer fruits and vegetables that are of similar size, color, and flavor.

Homeowners want lawns composed of grass plants with leaves of the same color and texture; they purchase Christmas trees with the shape and needle type that they remember from previous years.

When plants are grown from vegetatively propagated parts, the plants are not just genetically similar—they are identical. For better or worse, these plants will be resistant or susceptible to the same potential pathogens. Consider how many important plants are propagated vegetatively (i.e., not grown from seed): banana, potato, sweet potato, fruit trees (apple, cherry, citrus, peach, pear, plum), small fruits (brambles, grape, strawberry), nearly all greenhouse flower crops (carnation, chrysanthemum, poinsettia), and many woody ornamentals.

People affect the susceptibility of plants to disease by altering the plants themselves and by altering how they are grown. By developing genetically uniform crops and growing them in monocultures, sometimes over large areas, people create a situation conducive to epidemics. In addition, when people move plants out of their native environments, the plants are likely to encounter new pathogens to which they have little or no resistance.

A classic example of the dangers of genetic uniformity is seen in the late blight epidemic of potatoes in Ireland in the mid-1800s. Many poor Irish tenant farmers and their families subsisted mainly or entirely on potatoes. One cultivar, called Lumper, was very popular because of its high yields. However, the vegetatively propagated Lumper plants were particularly susceptible to the late blight pathogen, *Phytophthora infestans*, and their genetic uniformity was one significant cause of the epidemic and resulting famine (Figure 10.13).

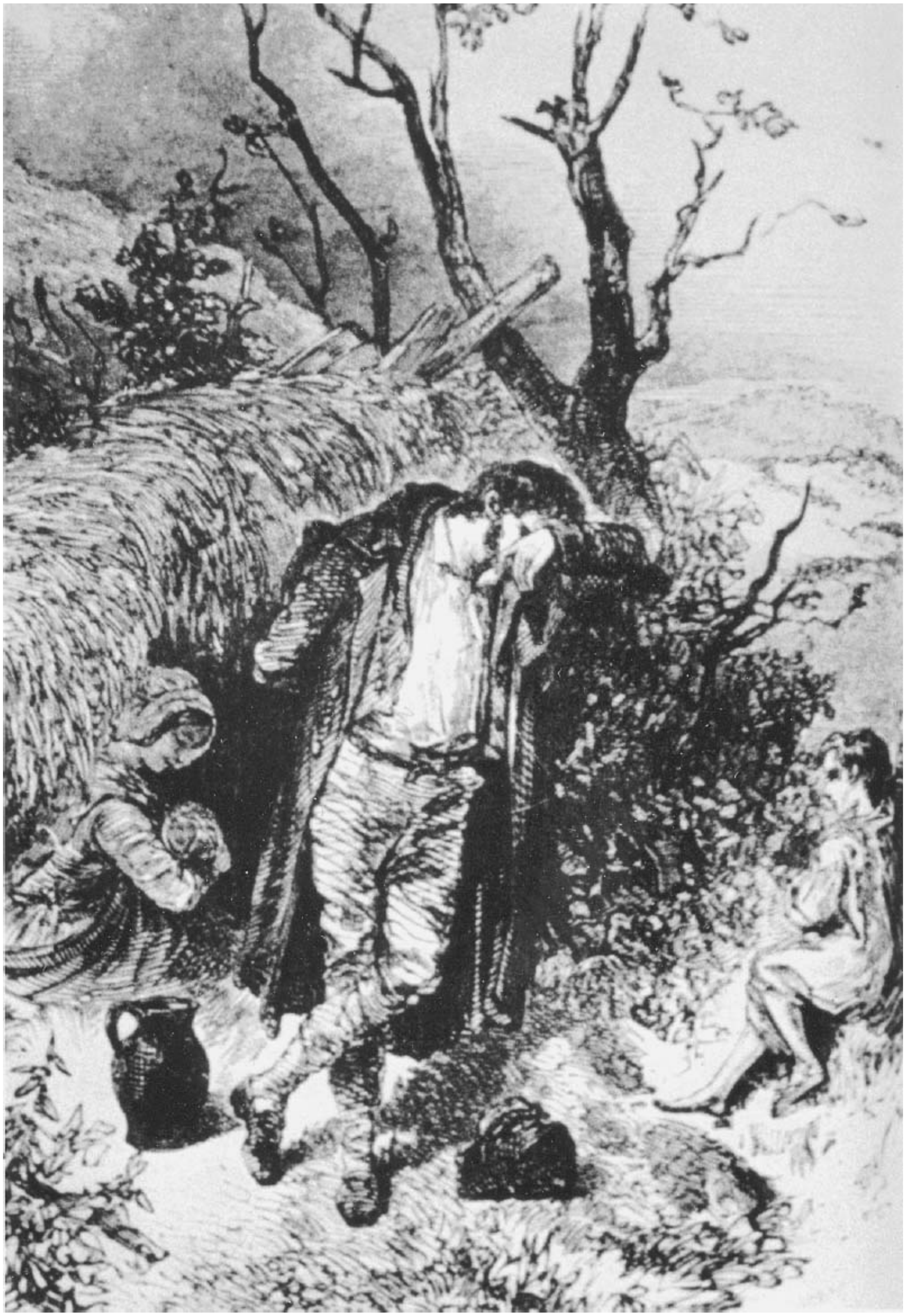


Figure 10.13. "After the Eviction." An Irish family evicted from their home because of failure to pay rent during the potato famine of the mid-1800s.

How do people affect the environment of plants in ways that increase disease?

Location

Not only do people plant large expanses of a genetically uniform plant species in agriculture, but they often plant them in the same locations year after year. This is sometimes unavoidable, as in the case of perennial plantings such as apple orchards, strawberry beds, golf courses, or lawns. Pathogen populations may increase over time until disease levels cause effects such as yield losses that are no longer acceptable, and then the plants must be replaced.

Planting choice in annual crops is often determined by economic factors such as the value of the crop, available markets, or the need for specialized and expensive planting and/or harvesting equipment for each type of crop. Planting the same annual crop in the same location for several years creates a situation similar to that in perennial crops and allows an increase in pathogen populations.

During the early 1900s, soybeans used for forage or green manure were grown in many fields across the United States every three or four years. This changed during World War II, however, when the demand for vegetable oil skyrocketed, and millions of acres of soybeans were planted for oil. To maximize production, soybeans were often grown in the same fields year after year. Growing the crop in the same locations allowed populations of soybean cyst nematode (*Heterodera glycines*) to increase each year, rather than declining as they had previously in years when nonhosts were grown. Eventually, the high nematode populations led to obvious chlorosis, stunting, and even death of soybean plants, although crop yields had actually been adversely affected even before symptoms were seen. Crop rotation remains one of the most effective management strategies for soybean cyst nematode today.

Water Management

Irrigation allows people to grow crops where they would not normally grow. It also can provide a reliable supply of water between rainfalls, which can increase yields or maintain attractive landscape plants and lawns. However, irrigation that increases leaf wetness (Figure 10.14) can exacerbate foliar diseases caused by fungi and bacteria, both of which require leaf wetness for infection. Water disseminates spores and bacteria. Sticky spores and bacteria are commonly distributed in water droplets. Even powdery spores can be distributed short distances in a water droplet because of their large surface area relative to their volume.

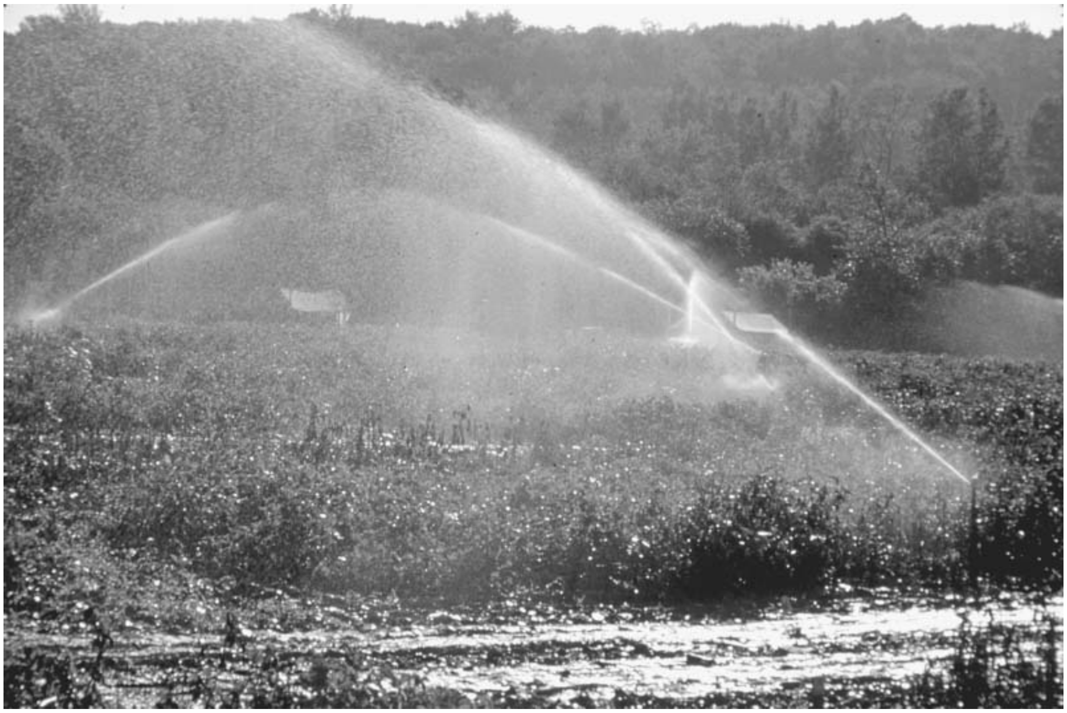


Figure 10.14. Irrigation can cause prolonged leaf wetness, which increases many diseases.

Verticillium wilt is a problem in the temperate regions of the world, especially where irrigation is used. Verticillium wilt of potato occurs wherever potatoes are grown and is most severe in irrigated fields, especially in hot climates. Yields of potatoes grown in *Verticillium*-infested soils may be 30–50% lower than yields on noninfested land. Overwatering, especially early in the growing season, results in increased infection and increased disease severity. Limiting the amount of water applied to a potato field can reduce the severity of Verticillium wilt.

DISEASE CLASSIC

Verticillium Wilt

Hosts over 200 dicotyledonous species, including herbaceous annuals (e.g., potato), perennials (e.g., peppermint), and woody species (e.g., maple)

Pathogen *Verticillium dahliae* (an ascomycete)

Symptoms and Signs Typical symptoms include foliar chlorosis and necrosis and stem and root vascular discoloration. Wilting occurs on warm, sunny days. Small, black microsclerotia are formed in dying host tissue and can be seen with a hand lens. The fungus is named for its verticels, which are white whorls of condiophores with masses of tiny conidia at each tip, observable with a microscope.

Disease Cycle Root exudates stimulate microsclerotia in the soil to germinate, and the fungus directly penetrates root tips. The fungus colonizes the root cortex and invades the xylem vessels, where conidia are formed. The conidia are transported to the rest of the plant through the xylem, and the vascular system becomes plugged, leading to wilt and chlorosis symptoms. As the host plant senesces, the fungus produces microsclerotia, which are released into the soil as the plant decomposes.

Management Soil should be tested for the presence of *Verticillium* before susceptible crops are planted. The number of microsclerotia can be somewhat reduced by crop rotation and, expensively, by soil fumigation. Planting pathogen-free stock, applying optimal levels of fertilizer, limiting the amount of irrigation, and burning crop residue at the end of the season also can help manage the disease. Cultivars of some host plants with good to moderate resistance are available.

Significance Verticillium wilt affects over 200 plant species in the temperate areas of the world. It reduces the yields of annuals and of herbaceous and woody perennials, including important food crops such as potato.

Plant Disease Profiles: Verticillium wilt

Water that is used to apply pesticides also can spread pathogens. For example, greenhouse-grown geraniums that received an application of an insecticide subsequently developed a rust epidemic because the water in the insecticide spray helped distribute rust spores throughout the planting.

Run-off water from irrigated plants can carry spores and other propagules to the soil, where they may survive and infect subsequent plantings. Irrigation ponds are a common source of *Pythium* and *Phytophthora*. Water traveling in irrigation canals and ditches may move soil and pathogens from farm to farm. When the new races of the Fusarium wilt pathogen (*Fusarium oxysporum* f. sp. *cubense*) responsible for Panama disease of bananas spread rapidly in Taiwan, it was discovered that farmers were disposing of infected corms in irrigation ditches.

Fertilizers and Other Inputs

Many foliar diseases can be categorized as low-nitrogen or high-nitrogen diseases. Plants with lush foliage due to nitrogen fertilization are more susceptible to pathogens that infect such tissue more easily. Other pathogens initially invade yellowed, senescent tissue, so low-fertility plants are more susceptible. When fertilizer is applied in relatively large, infrequent doses, plants alternate between being overfertilized and underfertilized and thus become susceptible to different diseases throughout the growing season. Turfgrass managers sometimes use a commonly occurring, low-nitrogen fungal disease called dollar spot (*Sclerotinia homoeocarpa*) as an indicator that the turf requires some nitrogen fertilizer. Homeowners who try to win the “spring green-up” competition in their neighborhood may lose their Kentucky bluegrass to the fungal disease melting-out (*Bipolaris* spp. and *Drechslera* spp.) if the lush leaf blades are exposed to a rainy week or two in the spring. A number of herbicides cause stress in desired plants as well as in the weeds they are designed to manage. This can leave plants more susceptible to disease.

Other Cultural Practices

No one would expect a surgeon to go from patient to patient with the same scalpel, but many plants are managed by shaping and pruning over the course of the season using the same tools for each cut and, in landscapes, for a variety of plants. Various tools and machines create wounds and distribute pathogens to the wound sites. This is especially true when maintenance is accomplished in the early morning when the plants are wet with dew.

Golf course putting greens often are mowed in the early morning. In many cases, dew is removed from the turf surface, which reduces fungal activity. However, if a disease such as anthracnose is present, mowers carry large numbers of sticky conidia to the cut surfaces of grass plants, and disease occurrence can follow mowing patterns from green to green.

Gray mold, caused by the fungus *Botrytis cinerea*, is a common problem on greenhouse crops as well as many herbaceous ornamental plants and many outdoor flower and vegetable crops.

The disease gets its name from the fuzzy, gray mycelium that grows on the surface of infected plants in humid conditions. The pathogen enters through wounds and infects very young or senescent tissue. Cool temperatures and high humidity (93–100%) favor its growth. Integrated management practices for this disease include careful handling of plants to avoid wounding and use of a variety of other cultural recommendations: reduce greenhouse humidity to avoid condensation (e.g., increase ventilation, avoid watering late in the day), increase spacing between plants (to allow light to penetrate to lower leaves and to enhance drying), supply sufficient nitrogen fertilizer to avoid yellowing of lower leaves, and remove dead and dying flowers and leaves where *Botrytis* can get established.

DISEASE CLASSIC

Gray Mold

Hosts a wide range of ornamentals (herbaceous annuals and perennials, shrubs, and trees), small fruits and vegetables; the disease is particularly important on nursery and greenhouse crops

Pathogen *Botrytis cinerea* (ascomycete)

Symptoms and Signs Symptoms can vary widely depending on the host. In general, leaves develop water-soaked spots that become gray to brown as they enlarge, sometimes forming concentric ring patterns. On flower petals, water-soaked to brown flecks develop into large tan to brown areas. Buds, stems, and crowns can also become infected, leading to blight, cankers, and even plant death.

Disease Cycle *Botrytis cinerea* is a weak pathogen and usually will not infect healthy, undamaged plants. The mycelium survives on dead plant material as a saprophyte and infects tissue that is wounded or senescing. *B. cinerea* is sometimes a damping-off pathogen. Under cool, humid conditions, a grayish web of mycelium grows over the infected tissues. Conidiophores develop in this web, and masses of conidia are released when infected plants are disturbed, mist is present, or the humidity rises rapidly. High humidity (93–100%) and low temperatures (7–16°C, or 45–60°F) favor disease development. Tiny sclerotia are common survival structures.

Management Sanitation is necessary to remove infected plant material, which should be burned or buried. Plants should be handled carefully to avoid wounding. Adequate ventilation, avoidance of overhead watering, and increased plant spacing will help keep foliage dry. Adequate nitrogen fertilizer should be applied.

Significance Gray mold is caused by one of the first genera of fungi to be scientifically described (1729). It is the most important greenhouse disease worldwide because of its wide host range and rapid dissemination by air and water.

Tillage (plowing) is one of the oldest agricultural practices. It has been performed by people, alone or with animals, for centuries, especially before farm machinery became available. Plant debris and stubble left after harvest are good survival sites for plant pathogens. Reduced tillage has been adopted in many areas to reduce soil erosion and to conserve soil water. This practice has increased the survival of primary inoculum for some important diseases such as gray leaf spot of corn (*Cercospora zeae-maydis*) and Fusarium head blight (scab) of wheat.

Plowing also is used to manage weeds, as are other methods, including herbicides and roguing. The incidences of many diseases, including those caused by pathogens with wide host ranges such as

Cucumber mosaic virus, root-knot nematodes (*Meloidogyne* spp.), *Tomato spotted wilt virus*, and *Verticillium*, increase when weed hosts are present.

People affect the environment of plants by

- selecting the location in which the plants are grown
- providing water for the plants
- providing nutrients (fertilizer) for the plants
- maintaining the plants with tools and machines

Climate Change and Plant Diseases

Although there is much debate about the impact of climate changes around the globe, there is general agreement that climate changes affect plants in both agricultural and natural ecosystems. The USDA has created its first new Plant Zone Hardiness Map since 1990, which delineates areas where different plant species are likely to survive. The new map is based on 30 years of weather data, which reveal that low temperatures during the winters have increased in many parts of the United States. A trend of increasing global temperature could impact plant disease epidemics in many ways. Warmer winters could mean increased disease severity through greater survival of and earlier infection by pathogens, such as was predicted in models for Phoma stem canker on oilseed rape in the United Kingdom. Warmer winters also may affect survival of pathogen vectors. Higher levels of CO_2 in the atmosphere could lengthen growing seasons and increase crop yields, and also could influence plant diseases. For example, Septoria brown spot was more severe on soybeans grown under elevated CO_2, but levels of downy mildew decreased in the same experiments. Research is just beginning on the potential impacts of climate change on plant disease epidemics.

People affect the environment in which plants grow in several ways. We may select locations, fertilization schemes, or watering schedules that increase the susceptibility of plants to disease. In addition, many of our cultural practices create wounds on plants that can serve as entry points for pathogens or can affect the survival of inoculum or alternative hosts.

How do people affect the ability of pathogens to cause disease?

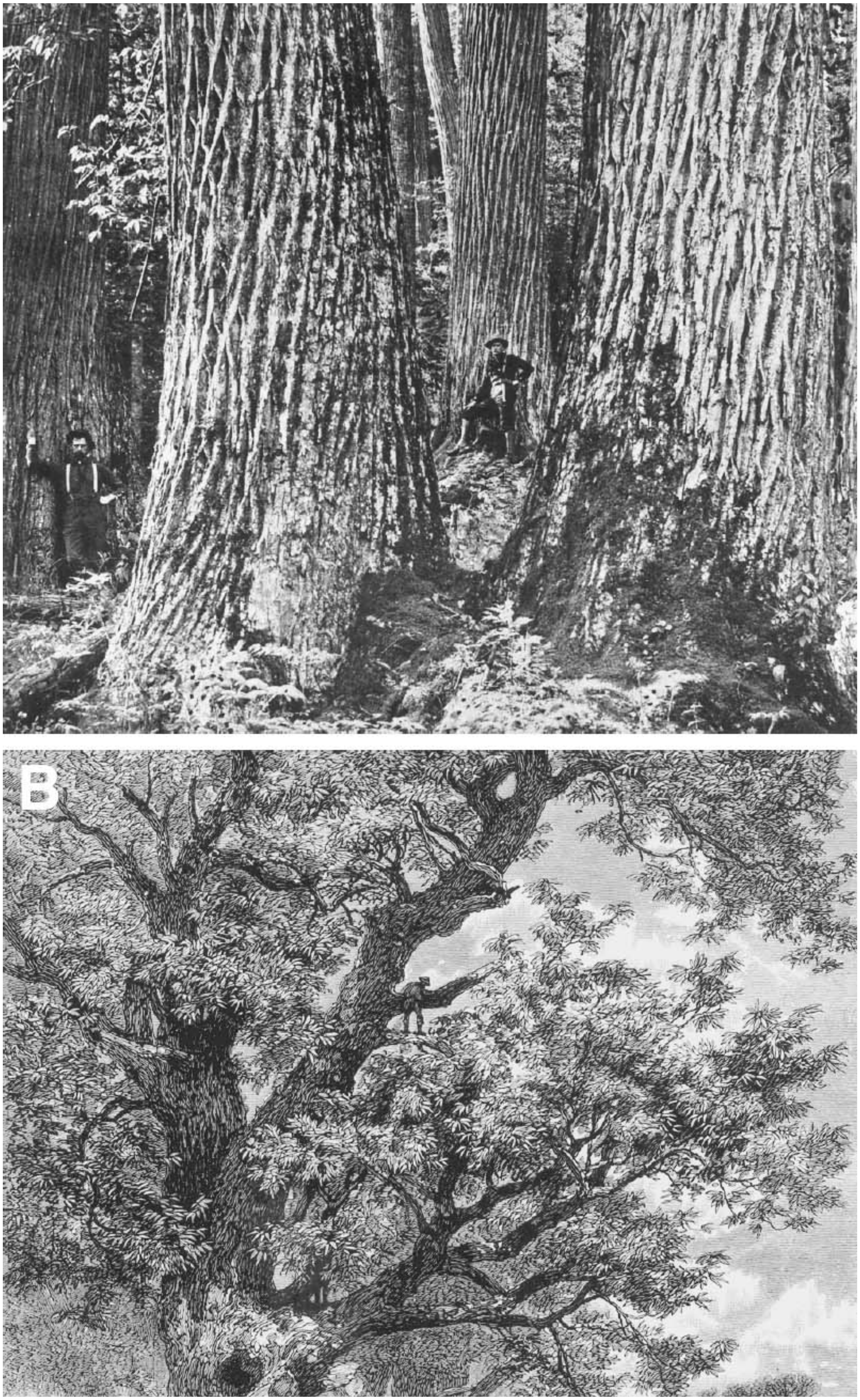
Introduced Pathogens

Unfortunately, when people transported plant species around the world, they also moved some of our most devastating pests and pathogens. Remember, however, that the germ theory of disease was not widely accepted until the mid-1800s, following the Irish potato famine, so for hundreds of years, people were not concerned about the potential introduction of new pathogens. As a result, a number of exotic pathogens have caused devastating epidemics after their unintentional introductions into new geographic areas. Accidental pathogen introductions continued to occur, even after the germ theory was accepted.

Several tree diseases became epidemic when an exotic pathogen was introduced to a native species. The impact of imported pathogens on trees is more obvious because their life span is so long, unlike annual field crops such as peanuts or wheat, even though these, too, are vulnerable.

The American chestnut was a dominant tree species from Maine to Georgia (Figure 10.15) until the introduction of the chestnut blight fungus (*Cryphonectria parasitica*) into New York on imported Asian chestnuts in 1904. Airborne dispersal of spores throughout the chestnut's natural range in the Appalachians rapidly destroyed this important species. By 1923, the disease had invaded 80% of the chestnut's range, and by the 1950s, 80% of the chestnut trees had died. Today, small shrublike trees grow from the roots of these former giants, but they survive only a few years before succumbing to the disease.

> Will the blight end the chestnut?
> The farmers rather guess not.
> It keeps smoldering at the roots
> And sending up new shoots
> Till another parasite
> Shall come to end the blight.
>
> —Robert Frost, "Evil Tendencies Cancel"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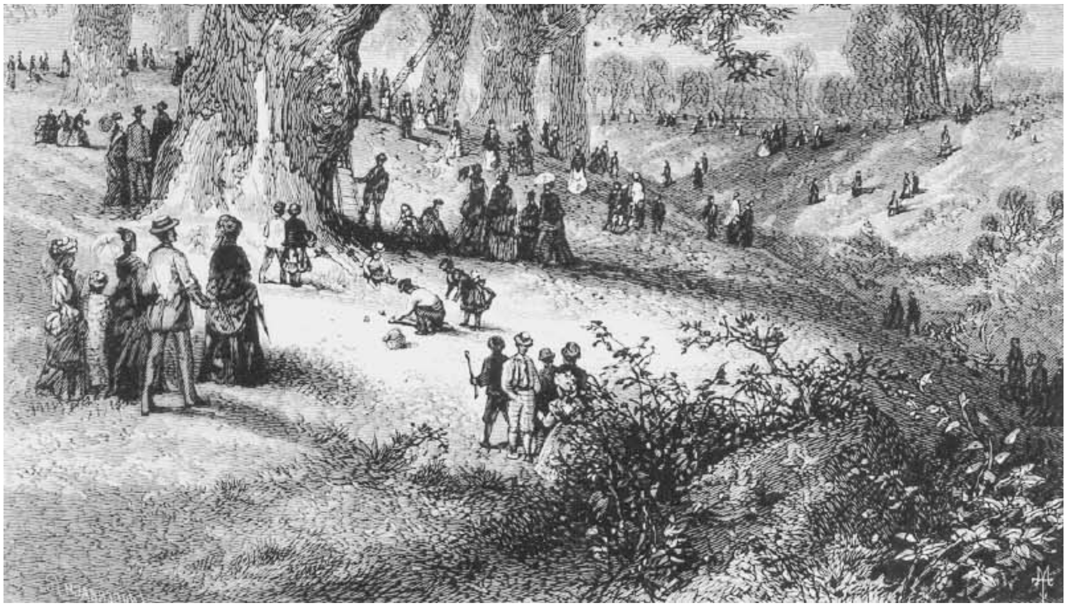

Figure 10.15. Chestnut trees in (A) the Appalachian forest and (B) a park in Philadelphia before chestnut blight had occurred in the United States.

Dutch elm disease was introduced into the United States on elm logs imported for furniture veneer in the 1920s. The logs contained both the causal fungus, *Ophiostoma ulmi*, and an efficient vector, the European elm bark beetle, *Scolytus multistriatus*. Monoculture plantings of elms lining city streets ensured the distribution of this vascular wilt (xyleminvading) fungus through root grafts. Over 40 million valuable shade trees have already been killed in this ongoing epidemic.

White pine blister rust (*Cronartium ribicola*) was introduced to the eastern United States and to western Canada around 1900 on pine seedlings imported from Europe for reforestation programs. By the mid-1900s, millions of white pines, highly prized for their lumber, were dead or dying.

DISEASE CLASSIC

White Pine Blister Rust

Hosts white pines (aecial hosts); currant, gooseberry (*Ribes* spp.) (telial hosts)

Pathogen *Cronartium ribicola* (a basidiomycete)

Symptoms and Signs In pine, the needles are infected first. Spindle-shaped swellings develop on infected branches and become cankers that exude large amounts of resin and can eventually girdle and kill the branch. Signs on pine include honey-colored droplets of pycniospores at canker margins in the spring, followed by masses of yellow-orange aeciospores, which erupt through the bark of the cankers. Symptoms on currants and gooseberries are chlorotic spots on the upper surface of leaves. Signs on these hosts are orange pustules containing urediniospores that develop on the underside of leaves. Later, brown, hairlike telial columns develop.

Disease Cycle Airborne basidiospores initiate infection through pine needle stomata in the fall. The fungus grows from the needles into the branches, where pycnia develop at canker margins in the spring after 1 or 2 years. Aeciospores, produced later in the spring, are airborne to *Ribes* spp., the telial hosts. Urediniospores produced on *Ribes* spp. can infect other currant or gooseberry plants. Later, teliospores form and germinate in the fall to produce basidiospores that can infect pine.

Management Pruning diseased branches of pines is somewhat effective but also is labor-intensive and expensive. The most common current management strategies are the destruction of localized susceptible *Ribes* spp.; avoiding planting in low areas where cool, moist air settles; and planting resistant pines, if available.

Significance White pine blister rust was introduced into the United States around 1900 on seedlings grown in European nurseries and imported for reforestation. The discovery of the disease led to the first U.S. quarantine law in 1912. Despite this law and others that authorized the destruction of *Ribes* spp., the rust was established throughout the major white pine regions of North America by 1950.

Plant Disease Profiles: White pine blister rust

The pathogens responsible for dogwood anthracnose (*Discula destructiva*) in the southeastern United States and Ramorum blight (sudden oak death) (*Phytophthora ramorum*) in California are believed to be more recent introductions of exotic species to the United States. Since the 1970s, large numbers of dogwood trees in both native forests and ornamental plantings have died from the exotic anthracnose fungus. The loss of trees to *P. ramorum* began around 1996 and has spread rapidly through coastal California (Figure 10.16). The wide host range and ease of transport on potted nursery stock make this disease particularly threatening.

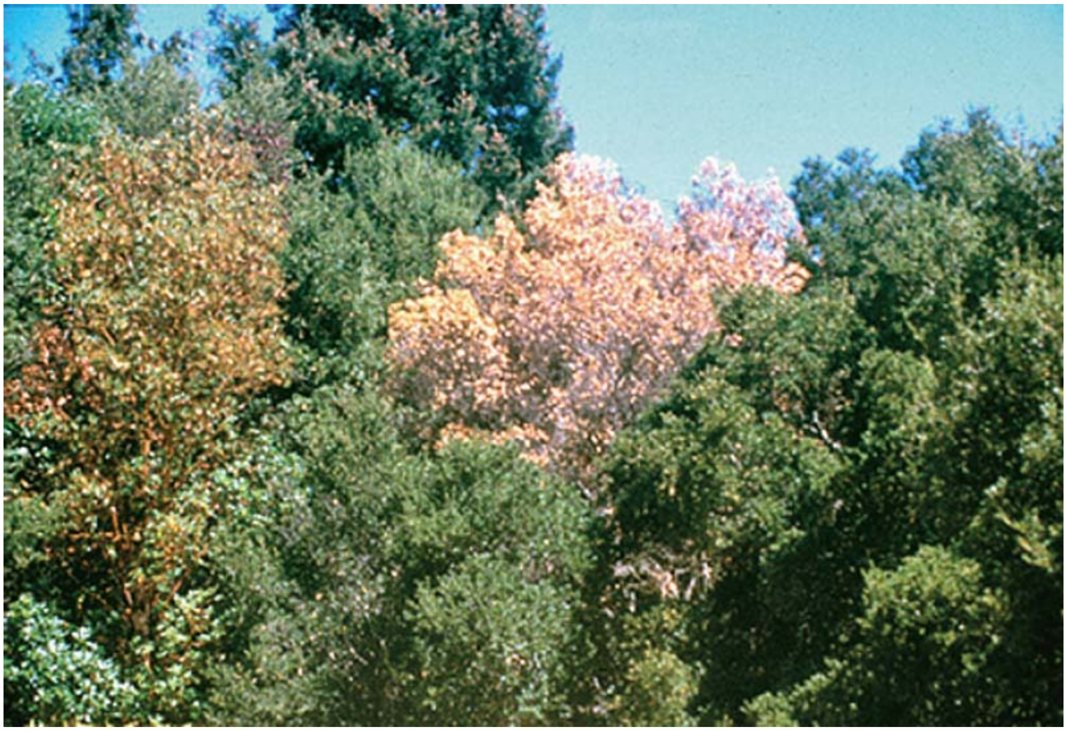

Figure 10.16. Ramorum blight, also known as sudden oak death, caused by *Phytophthora ramorum*, in California.

Of course, pathogens native to the United States have been responsible for epidemics in other parts of the world. The pine wood nematode (*Bursaphelenchus xylophilus*) that causes pine wilt disease is native to North America. Asian and European pines do not have much resistance to the nematode and can die in a single season following infection. The introduction of this nematode into Japan has resulted in 50% losses in many pine forests. Timber and wood chips must be fumigated or heat-treated before export from North America to Europe or Asia because of this pathogen.

In some cases, plants from the same center of origin as a pathogen become susceptible to it when they are grown for long periods of time in locations where the pathogen is absent. When the pathogen is later introduced to these locations, devastating epidemics can result.

Potatoes, which originated in the highlands of the Andes in South America, were slow to be accepted in Europe as a food crop, but they were widely planted and eaten by the lower classes for nearly 300 years in the absence of late blight. It is not clear exactly when the pathogen, *Phytophthora infestans*, was reintroduced to this crop in North America and Europe, but catastrophic epidemics occurred in the rainy 1840s and continue to this day.

Both citrus plants and the citrus canker bacteria (*Xanthomonas axonopodis* pv. *citri* and *X. axonopodis* pv. *aurantifolii*) are native to Asia. Nearly all citrus is vegetatively propagated, and nearly all cultivated citrus is susceptible to this disease, especially grapefruit and sweet orange. The disease is endemic in many citrus-growing countries in Asia, the Middle East, and South America. Citrus trees in the southeastern United States were isolated from the citrus canker pathogens for decades until 1915, when citrus canker was first reported. After a concerted effort to cut and burn infected trees, citrus canker was long offered as the only example of the successful eradication of a pathogen (Figure 10.17). However, in 1985, the disease was again reported in Florida, and a controversial inspection and tree-removal campaign to eradicate the disease was implemented. This campaign ended in 2006 after several hurricanes disseminated the bacteria across the entire state of Florida.

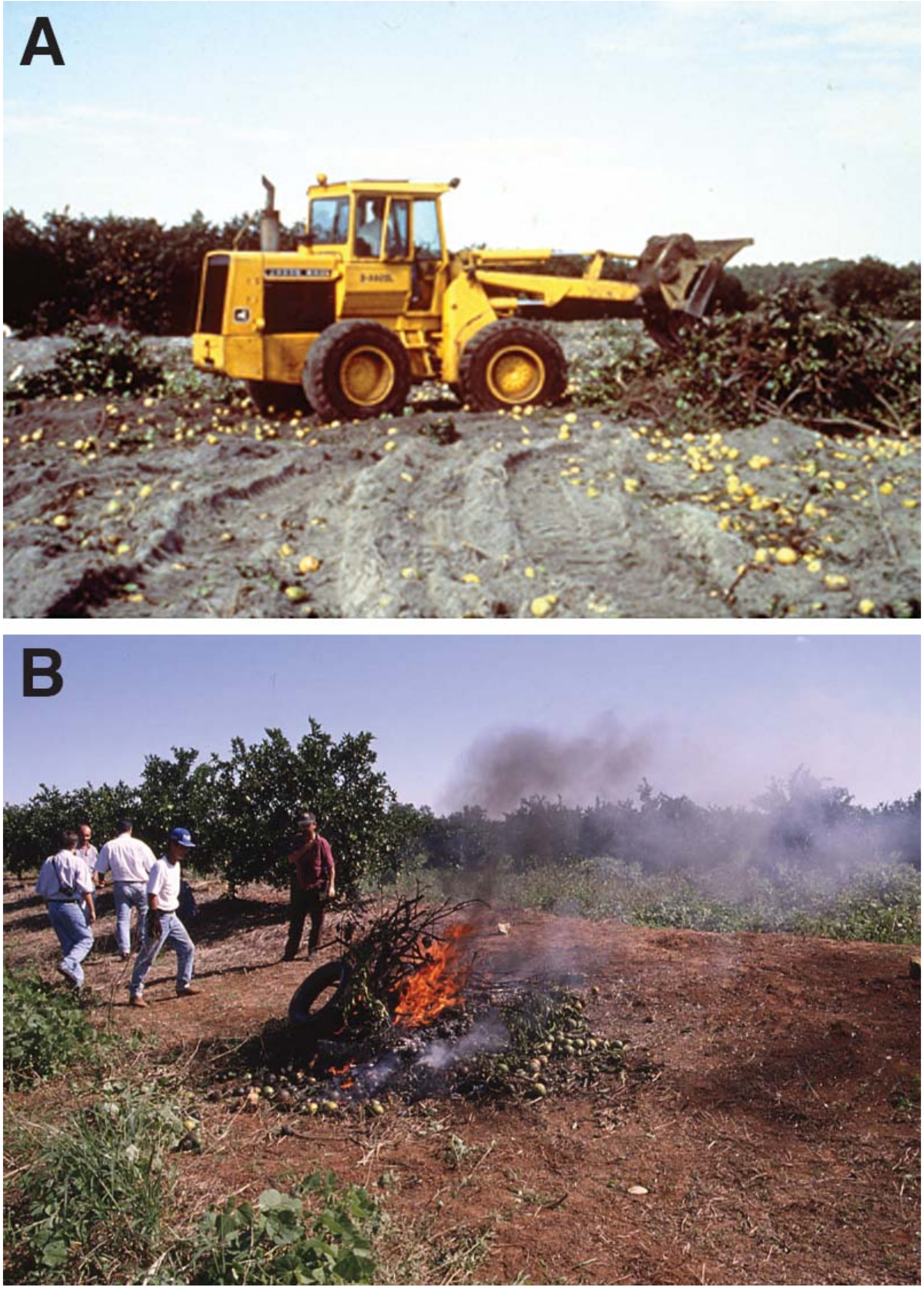

Figure 10.17. Citrus trees infected with citrus canker destroyed by plowing (A) and burning (B).

In 2004, soybean rust was confirmed in many soybean-growing states of the United States. Both soybeans and their rust pathogens are native to Asia, but rust had been excluded by quarantines from the valuable U.S. soybean crop. Breeders have selected new soybean cultivars for genetic attributes unrelated to rust resistance, and so to date no resistance occurs in the germplasm of U.S.-grown soybeans. The impact of this disease on world soybean production remains to be seen.

Introduced Vectors

Vectors That Have Been Moved to New Areas and Increased Disease

Vector	Pathogen
European elm bark beetle	*Ophiostoma ulmi*
glassy-winged sharpshooter	*Xylella fastidiosa*
western flower thrips	tospoviruses
whitefly	geminiviruses

Many pathogens cannot cause serious losses without a vector to move them from plant to plant. The introduction of the vector has been a key factor in the increased importance of a number of diseases (Figure 10.18). As described above, the well-known Dutch elm disease epidemic resulted from the introduction of both the fungal pathogen and an efficient new vector, the European bark beetle. This same vector has been introduced into Australia, where immigrants from England have planted millions of elm trees, although the fungal pathogen has, so far, not been introduced.

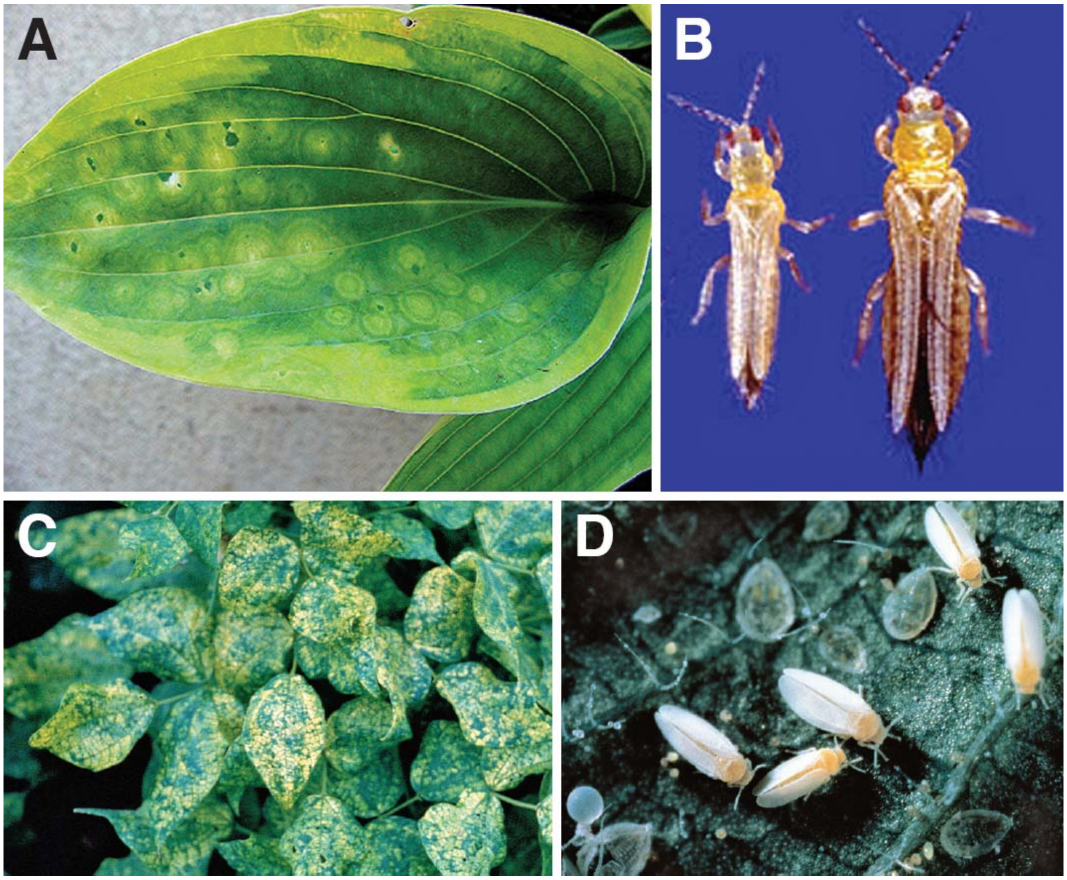

Figure 10.18. Tospoviruses, such as *Tomato spotted wilt virus*, which causes a disease of hosta (A), are transmitted by thrips (B). Some geminiviruses, such as *Bean golden mosaic virus* (C), are transmitted by whiteflies (D).

Since the mid-1980s, geminiviruses have caused tremendous losses in cucurbits, legumes, and especially tomatoes because of the introduction of a new whitefly biotype or species (*Bemisia tabaci*) to the tropical and subtropical regions of the Western Hemisphere. During the same period, the western flower thrips (*Frankliniella occidentalis*) has become an important worldwide vector of most tospoviruses, such as *Tomato spotted wilt virus*, and affects production of many flower and vegetable crops in greenhouses and outdoors. The glassy-winged sharpshooter (*Homalodisca coagulata*) is an introduced vector of the fastidious bacterium *Xylella fastidiosa* in California. It can transmit this pathogen (which causes Pierce's disease in grapevines) to alfalfa, almond trees, citrus, and oleander.

Since it was first reported in the state in 1990, *H. coagulata* has rapidly spread throughout much of southern California.

Seedborne Pathogens

When seed is harvested from a diseased plant, it may carry pathogens. An infested seed carries the pathogen on its surface, and the germinating seedling is subject to infection. If the cotyledons or embryo within the seed are infected, the resulting plant will certainly be infected. For many centuries, farmers could only try to select healthy-looking seed for planting. Losses due to seedborne pathogens were widespread, especially following years in which the mother plants were severely diseased. Smut diseases of cereal crops and bacterial and viral diseases of flowers and vegetables were especially common and difficult or impossible to manage once the seedling was infected. Today, many seed stocks are routinely tested for the presence of particularly damaging seedborne pathogens. Despite these precautions, some seedborne diseases continue to cause significant economic losses.

► **DID YOU KNOW?**

In some cases, the seedborne nature of pathogens was misinterpreted. For example, the wheat seed gall nematode, *Anguina tritici*, invades developing wheat seeds by swimming from the soil when young wheat plants are wet. When the resulting seed galls are cracked open in water, numerous nematodes will appear. Long ago, the appearance of the nematodes in the seeds was interpreted as evidence of spontaneous generation.

Examples of Currently Significant Seedborne Diseases

Disease	Host	Pathogen
bacterial speck	tomato	*Pseudomonas syringae* pv. *tomato*
bacterial spot	tomato	*Xanthomonas campestris* pv. *vesicatoria*
blackleg	brassica (crucifer) vegetable crops, oilseed crops (e.g., canola)	*Phoma lingam*
loose smut	cereals	*Ustilago* spp.
stinking smut (bunt)	cereals	*Tilletia* spp.

Vegetative Propagation

Vegetative propagation is even more likely than seed production to produce plants that carry pathogens to the next generation. Even when propagators try to choose plants that appear to be healthy, it is difficult to determine whether a plant may be infected by a virus, vascular wilt bacterium or fungus, fastidious bacterium, or other systemic pathogen. In the 1940s, chrysanthemum propagation was dominated by a few specialist propagators, who then shipped their plants throughout the world. Two systemic diseases, first Verticillium wilt and then chrysanthemum stunt, were spread in the mum cuttings and threatened the very existence of the chrysanthemum industry.

► DID YOU KNOW?

For centuries, farmers were aware that the yield of vegetatively propagated plants, such as potatoes, would decline after some generations. However, until the twentieth century, they were unaware that this was because viruses and other pathogens were accumulating in the tubers. The farmers speculated that the plants were becoming weak due to lack of sexual reproduction. Their theory seemed to be corroborated because the vigor and yield of plants were restored when true seed was used. Actually, the use of true seed was simply eliminating many of the tuber-borne pathogens responsible for the problem.

Detection of pathogens in relatively inactive vegetative parts, such as bulbs, corms, and tubers, is difficult. The propagative piece itself may be **infected** or **infested**, and, because they are produced in soil, they are often contaminated by pathogen-infested soil. People are likely to ship large quantities of these items for food and as propagative parts for new plantings of crops such as banana, potato, and tulip.

Ring rot of potatoes, a bacterial vascular wilt caused by *Clavibacter michiganensis* subsp. *sepedonicus*, can reduce yields by as much as 50%. The vascular rings of infected tubers are filled with ooze (Figure 10.19). When tubers used for planting are mechanically cut, a single infected, oozing tuber can potentially contaminate the cut surfaces of numerous tuber pieces. There is zero tolerance for this devastating disease in potato seed tuber production. If a single infected plant is found in a field, the harvested tubers from the entire field may be sold only as produce, not as seed tubers. Despite these precautions, the pathogen has still been moved around the world in seed potatoes, most recently to the United Kingdom.

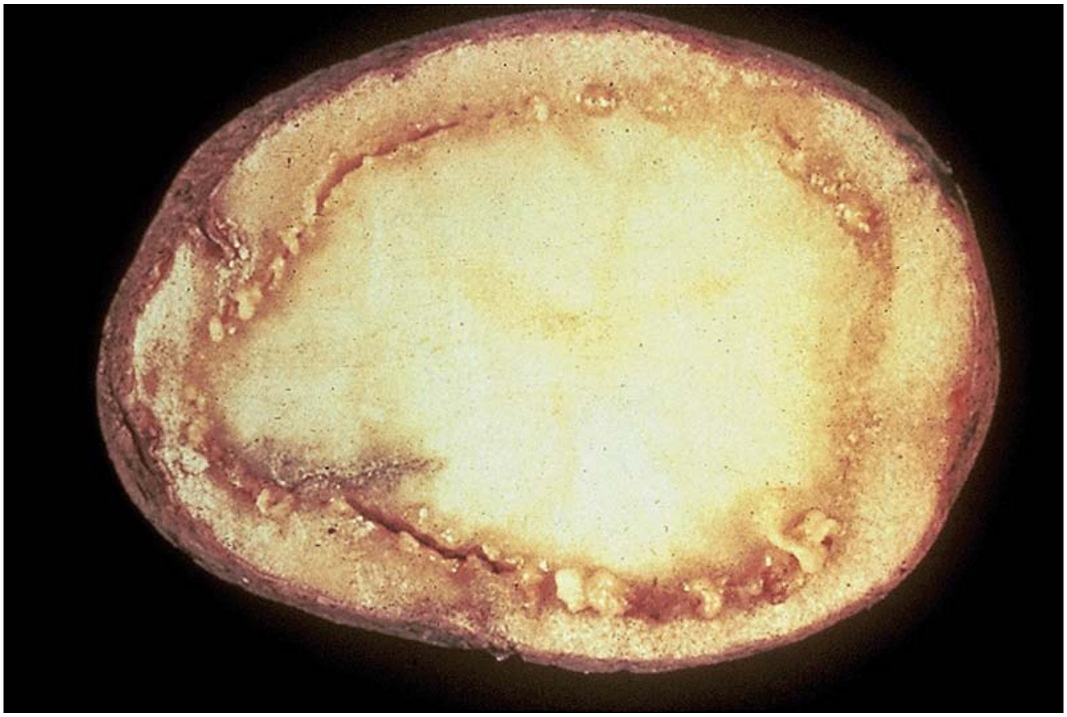

Figure 10.19. Ring rot of potato. Cutting tools can easily spread the bacterial ooze from a diseased tuber to the cut surfaces of seed tubers.

Although grapes are not a staple food, the wine produced from them is an economically important product. In the 1800s, the French wine industry was threatened by a series of pests and pathogens. One of these, the fungal pathogen *Plasmopara viticola*, which causes grape downy mildew, was introduced into France on rootstocks imported from the United States. Ironically, the rootstocks infected with downy mildew were imported because they were resistant to the aphidlike *Phylloxera* pests that were destroying the crop.

People have inadvertently moved many pathogens and their vectors from their native areas to new locations where they have caused epidemics in susceptible local plant populations.

These plants may never have encountered the pathogen before, or they may have been physically separated from it for a long period of time. People transport pathogens in or on seed, in vegetatively propagated crops, and in plant products to new locations, where epidemics can occur.

How do plant disease epidemics affect people?

Economic Effects

The most common impacts of plant disease epidemics on people today are probably economic. Losses caused by both biotic and abiotic disease agents mean lower yields and therefore reduced income for producers and restricted availability and therefore higher prices for consumers. The prices of plants or plant products (including food, feed, clothing, and building materials) may be higher. The fruits and vegetables that provide important nutrients are particularly vulnerable to diseases that can affect both their abundance and their quality. For relatively affluent people, these economic effects may be unnoticed or just a minor nuisance. However, for those living at the margin, a reduction in farm income, a decrease in food availability or quality, or an increase in the price of a staple food can be disastrous.

Effects of Plant Disease Epidemics on People

- Yield losses cause reduced availability and increased prices (e.g., many diseases every year).
- Destruction of an ornamental plant reduces the beauty of our environment (e.g., Dutch elm disease).

- Destruction of a crop results in a change in agriculture (e.g., coffee rust).
- Loss of a critical food crop results in famine (e.g., Bengal famine, Irish potato famine).
- Postharvest losses affect food supplies between crops (e.g., rotten, moldy stored food).
- Mycotoxin contamination causes human or animal diseases (e.g., aflatoxin, ergot).

Such effects on producers were seen in an epidemic of southern corn leaf blight in the United States in 1970, when many family farms were lost. In 1995, losses to late blight of potato exceeded $30 million in the Pacific Northwest. A record epidemic of Fusarium head blight (scab) of wheat in North and South Dakota and Minnesota had an economic impact of more than $1 billion in 1993. Deleterious economic impacts on consumers are illustrated in historical epidemics, such as the Irish potato famine, when late blight prevented the tenant farmers from producing the potatoes needed to feed their families.

Because of the globalization of trade, pests and pathogens can affect the ability of a country to import or export crops and plants around the world or even to move them within its own borders. In 1996, Karnal bunt (*Tilletia indica*), a disease of wheat, was discovered in the southwestern United States, and the wheat crops in this area were put under immediate quarantine. This was a significant discovery because 21 countries have quarantines against Karnal bunt. If Karnal bunt is detected in a wheat crop, the wheat cannot be exported to those countries. The United States is the world’s largest wheat exporter, providing one-third of world wheat exports, valued at approximately $5 billion in fiscal year 1995.

The citrus canker quarantine in Florida limited the import of citrus fruits into the United States and the shipment of Florida citrus to other citrus-producing states. Europe and much of Asia require that wood products from North America undergo expensive heat or chemical treatments prior to export to prevent the import of pine wood nematodes.

► **DID YOU KNOW?**

In the United States alone, annual crop losses due to plant pathogens amount to $9.1 billion. Worldwide, plant diseases reduce crop productivity by 12% (Food and Agriculture Organization, 1993).

There are a few positive economic impacts of plant diseases today, such as edible, smutted corn sold as *cuitlacoche* or the desirable bushy shape of poinsettias due to a phytoplasma infection. Historically, one plant disease had a highly positive economic impact over a period of years. In the early 1600s, tulips that were infected by *Tulip breaking virus* (TBV) and produced “broken” (variegated) flowers were highly prized in the Netherlands (Figure 10.20). For several years, these tulips were used as money, and their value could double every few months. This “tulipomania” ended in February 1637 when the market for the bulbs crashed.

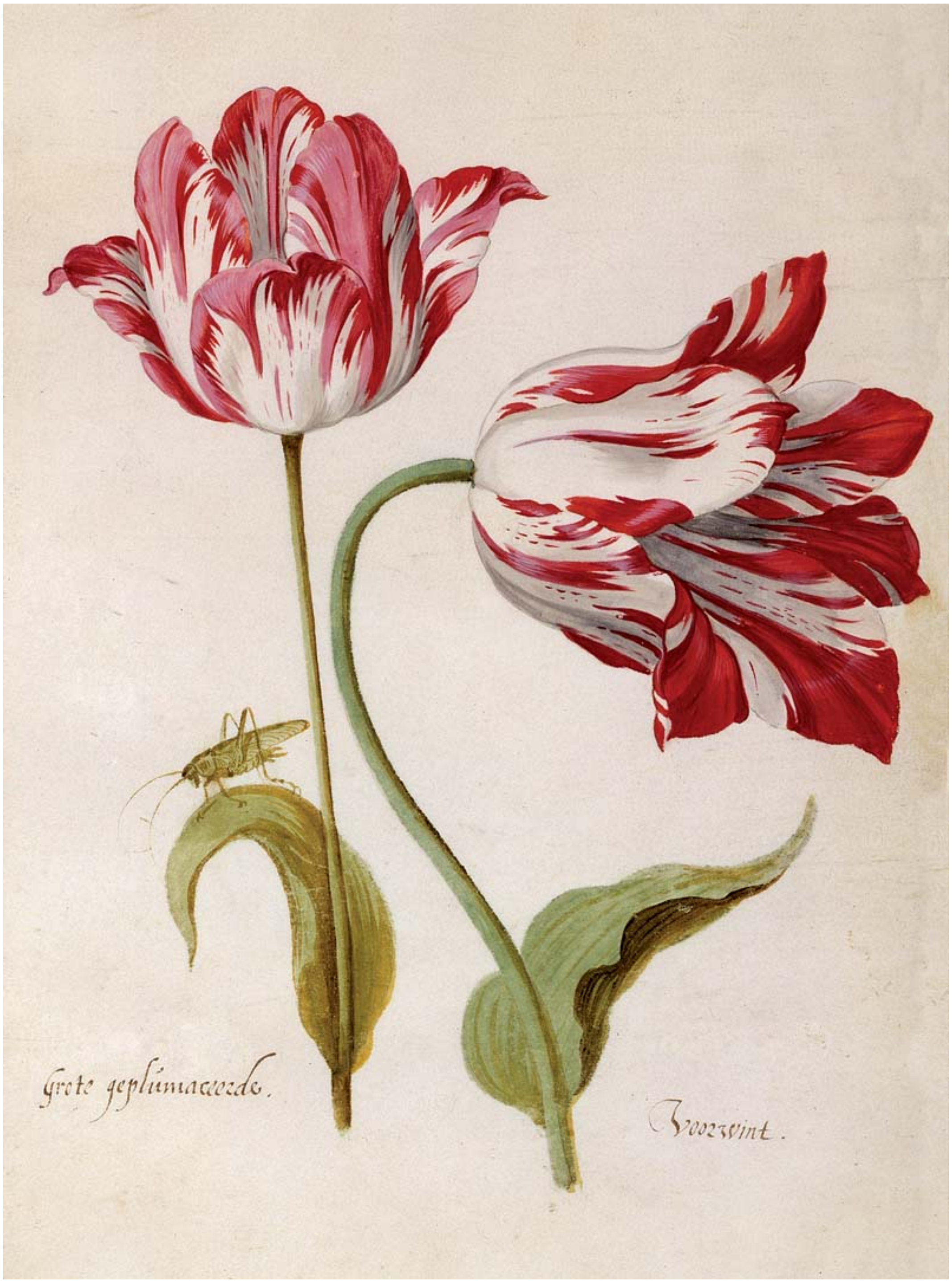
Grote geplúmaceerde.
Voorwint.

Figure 10.20. Tulips with color break in a painting by Dutch artist Jakob Marrel (1614–1681).

Aesthetic Losses

Although not often measured in monetary terms, plant diseases cause significant aesthetic losses when they reduce the beauty of plants or kill them entirely. Recent research indicates that the presence of plants in our environment has positive effects on our thoughts and actions. The loss of these plants to disease, such as the loss of millions of elm trees to Dutch elm disease, reducing the comfort and beauty of shady city streets, is therefore significant (Figure 10.21). Even small disease blemishes on flowers, fruits, ornamentals, and vegetables detract from their aesthetic value and reduce some of the pleasure we derive from them.

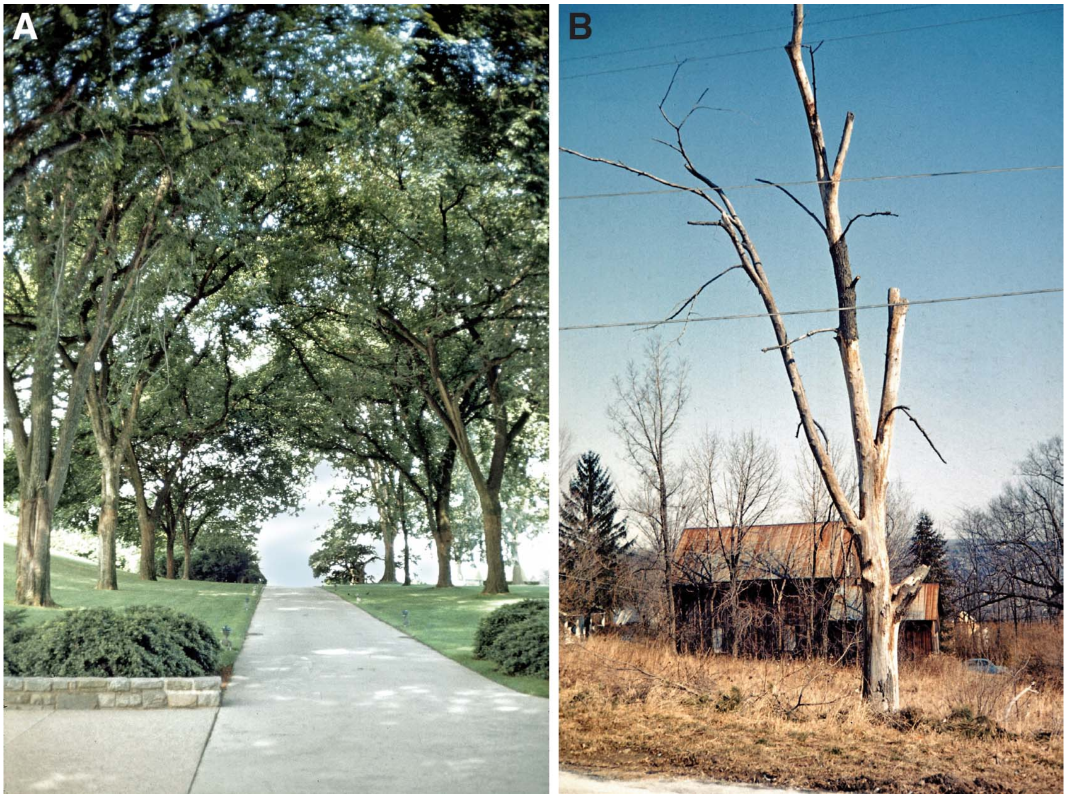

Figure 10.21. American elms (A) lined many streets in the United States before they were killed by Dutch elm disease (B).

Crop Production Changes

Plant diseases affect what plants we can grow and where we can grow them. The effects may be small, such as discovering that you cannot grow roses in an area of your yard where the soil is infested with the crown gall bacterium. Of greater significance is the fact that pears usually cannot be grown in humid climates of the United States because of fire blight. Plant diseases can even change the crop production practices of entire countries. The island of Ceylon (now Sri Lanka) changed from coffee plantations to tea plantations in just a few years after coffee rust destroyed the coffee trees.

Famine and Emigration

When plant diseases cause staple food supplies to become critically short, people face famine and may be forced to emigrate. An epidemic of brown spot (caused by the fungus *Bipolaris oryzae*) caused losses of 70% to more than 90% of the rice crop in the Bengal region of India in 1942, resulting in the Bengal famine that caused the death of an estimated 2 million Indians. The potato late blight epidemic resulted in the death or emigration to North America of millions of Irish in the mid-1800s. Unfortunately, diseases still cause famines today, particularly in developing countries.

Food Security and Food Safety

Many of the pathogens that reduce quality and yield in the field continue to rot our food after it is harvested. This reduces how long we can store fruits and vegetables that add variety and important nutrients to our diet. Grain and other seeds are easier to store than more perishable crops, such as fruits and vegetables, and therefore are relied on for food security between crops and when crops fail (Figure 10.22). People who live in relative wealth can afford to reject moldy food, but poorer people often cannot afford this luxury.

Figure 10.22. Large quantities of food and feed (soybeans are shown here) must be stored after harvest.

One aspect of postharvest yield loss that is often overlooked is the fact that some fungi can produce toxic materials in food. Obviously rotten fruits and vegetables are usually too unappealing to eat, so they are discarded (Figure 10.23). Of more concern is moldy growth in stored grain and other seeds. Even a small pocket of moldy growth can result in high levels of **mycotoxins**. The mycotoxins may not alter the appearance, texture, taste, or smell of the food, but they definitely spoil it. Fungi in the genera *Aspergillus, Claviceps, Fusarium*, and *Penicillium* produce mycotoxins that are harmful to both animals and people (Figure 10.24). The Food and Agriculture Organization (FAO) estimates that 25% of the world's food crops are significantly contaminated by mycotoxins. Moldy grain, which we would avoid eating if given a choice, is usually processed into flour and other products. The fungal contamination may no longer be evident when people buy processed food, but the mycotoxins may remain.

Some Fungi That Produce Mycotoxins

Aspergillus flavus	anatoxins
Claviceps purpurea	alkaloids
Fusarium spp.	fumonisins, trichothecenes, zearalenones
Penicillium spp.	patulin, rubratoxin

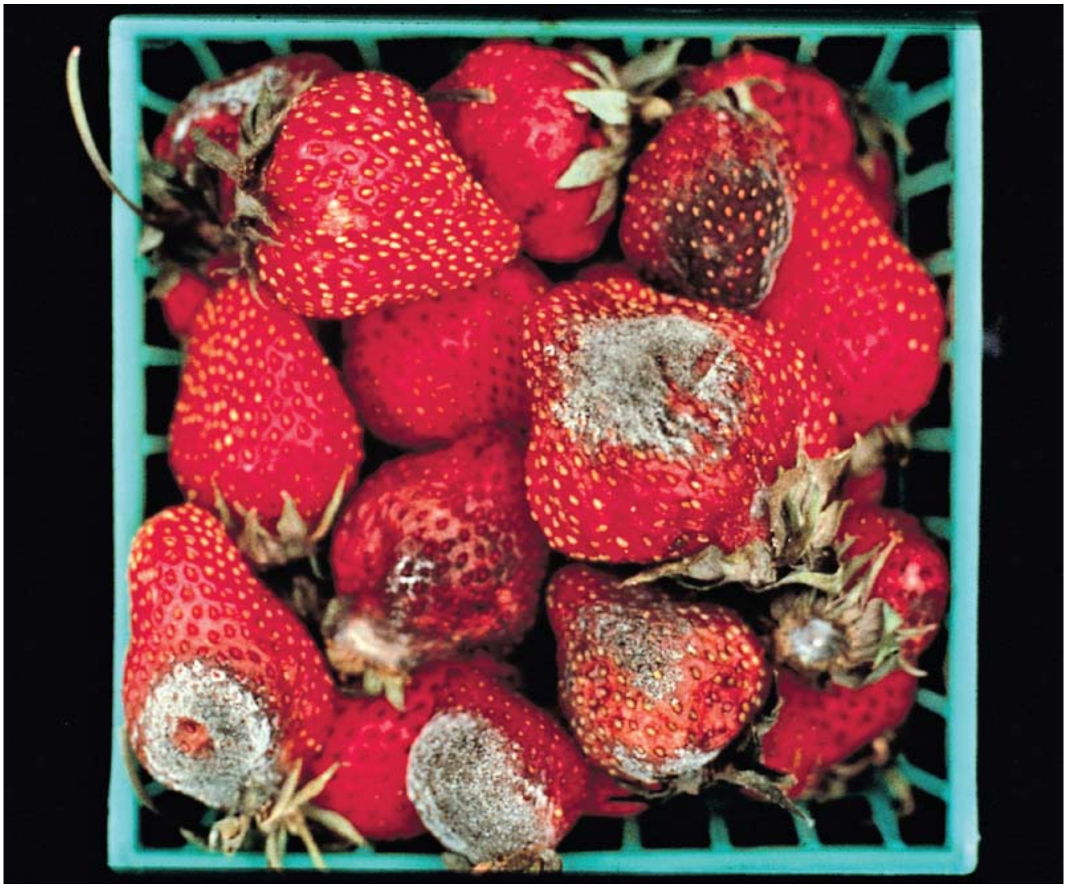

Figure 10.23. Strawberries that have rotted after harvest as a result of gray mold, caused by *Botrytis cinerea*.

► DID YOU KNOW?

Ergot is a disease of some grasses and cereals (especially rye) caused by *Claviceps* species; *ergot* is also the name given to the sclerotium, or resting structure, of the fungus. The sclerotia replace kernels of grain in heads produced from infected flowers. Mycotoxins produced by the fungus in ergot-contaminated grain cause ergotism, a disease characterized by hallucinations, burning skin, and gangrene. Thousands of people have died of ergotism. Mortality rates averaged 40% in some documented epidemics in the 1800s. Even after the cause of ergotism was known, many poor people did not have alternative food sources in years when ergot was severe in rye. Today, U.S. government standards prohibit the sale of grain for human consumption if it contains more than 0.3% ergot by weight.

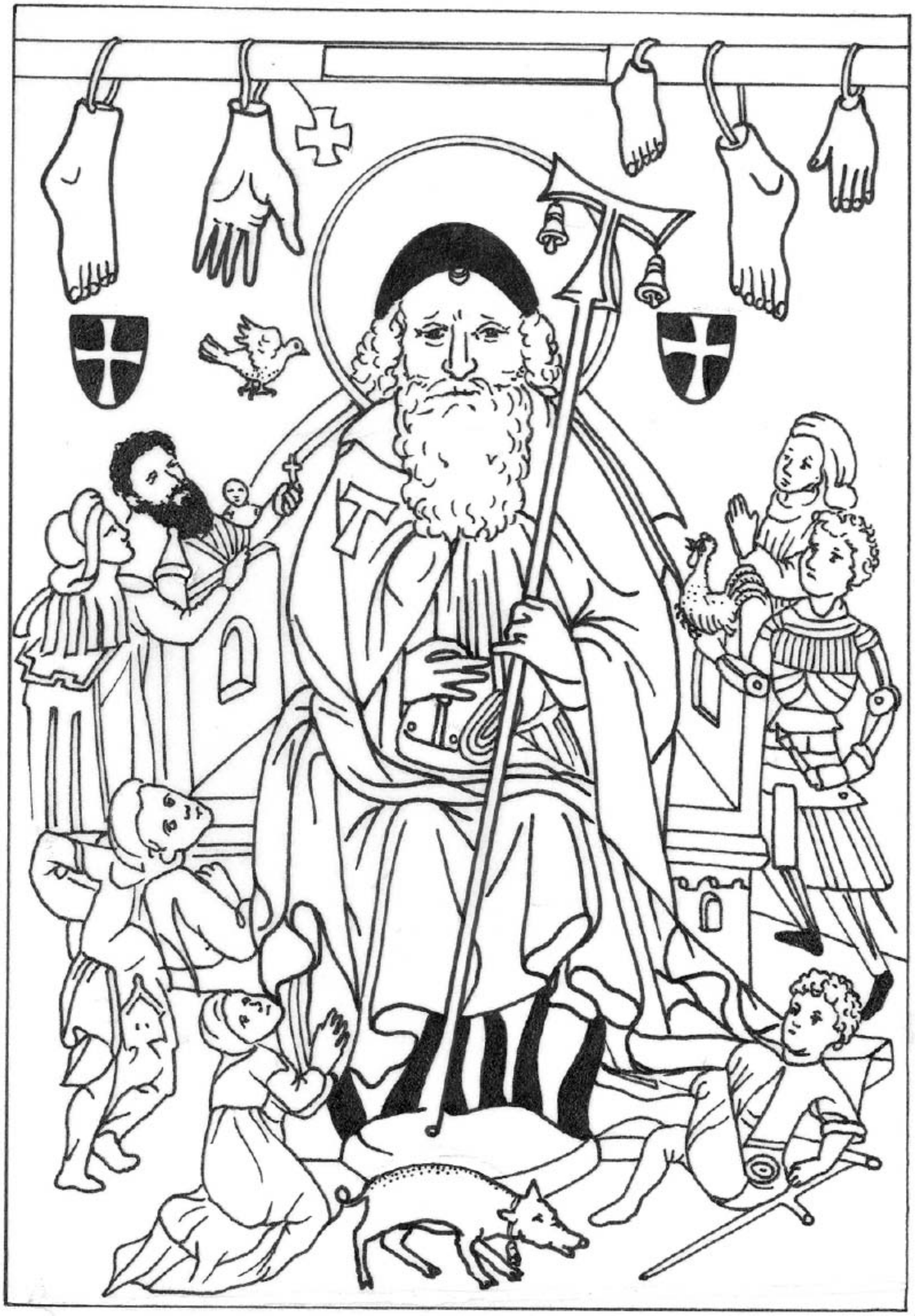

Figure 10.24. St. Anthony. Redrawn from a woodcut made in Germany about 1215. Note the hands and feet hanging on the top bar. Loss of extremities from gangrene was an important symptom of ergotism, also known as St. Anthony's fire, a disease caused by mycotoxin poisoning.

Some mycotoxins are known to be **carcinogens**, **immunosuppressants**, and/or **mutagens**. Increased incidences of certain diseases, such as liver cancer and esophageal cancer, have been found in populations that routinely eat foods containing mycotoxins. The likelihood of mycotoxin exposure remains greatest in developing countries, where the ingestion of moldy food is more common because food is more often in short supply and environmental conditions may favor fungal growth for much of the year.

► **DID YOU KNOW?**

In the 1960s, more than 100,000 turkeys died on farms in England from a new and unknown disease that was named "turkey X disease." Later, it was discovered that they had been fed peanut meal contaminated with anatoxin. This led to a greater awareness of the impact of mycotoxins on food safety for animals and humans. Birdseed used for outdoor bird feeders can become moldy and contaminated by mycotoxins in rainy weather, so feeders should be cleaned frequently in wet weather to protect wild birds from poisoning.

People are affected by plant disease epidemics every day—and have been for millennia. These effects can be relatively minor, such as an increase in the price of a luxury food item, or they can be devastating, such as the loss of a staple food. Plant diseases impact what crops we can grow, how much food or other products we harvest, how safe our food is to eat, and sometimes even whether we have food to eat at all.

Epidemics may force people to move to another place in order to survive.

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Questions

1. How do plant diseases caused by monocyclic pathogens differ from those caused by polycyclic pathogens? How do the strategies for management of epidemics caused by these two types of pathogens differ? Why are they different?

2. How would you create a prediction system for a monocyclic pathogen? How would a prediction system for a polycyclic pathogen be different? Why do you think that many plant managers continue to spray fungicides on a calendar schedule and ignore prediction systems?
3. List three ways that natural ecosystems differ from agricultural ecosystems. How does each item in your list impact plant disease epidemics, if at all?
4. Explain why plants that are growing near their center of origin are less likely to suffer devastating plant disease epidemics than plants that are growing where they are not native.
5. Explain two ways that people commonly influence the susceptibility of plants to disease. Give an example of a classic disease epidemic that was caused by each of your choices.
6. People can influence the environments in which plants grow in many different ways, some of which encourage disease and some of which prevent disease. Discuss an example of each situation.
7. There are many vectors of plant pathogens, including people! Describe two common ways that people move plant pathogens around the world.
8. Food safety has received a lot of publicity in recent years. Explain how some plant diseases reduce food safety and what we can do about this problem. In particular, how can you reduce mycotoxins in your diet?
9. How have you or someone you know personally been affected by a plant disease epidemic? Describe the epidemic and its consequences.

Challenge Questions

1. What is an example of a polycyclic pathogen that can be effectively managed by reducing the amount or efficacy of its

primary inoculum? What is an example of a polycyclic pathogen that must be managed by reducing the rate of infection? What are the most important environmental factors that affect the rate of infection for the polycyclic pathogen?
2. In what ways are the world's food and fiber crops vulnerable to bioterrorism?
3. What environmental factors have the most impact on aboveground plant diseases? Are the environmental factors that impact belowground plant diseases the same or different and why?

Supplementary Resources

Illustrated Glossary

Special Topic

Mycotoxins in crops: A threat to human and domestic animal health

Plant Disease Profiles

Citrus canker
Coffee rust
Downy mildew of grape
Dutch elm disease
Ergot
Fire blight of apple and pear
Late blight of potato and tomato
Pine wilt disease
Soybean cyst nematode disease
Stem rust of wheat

- Tomato spotted wilt
- Verticillium wilt
- White pine blister rust

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

- carcinogen
- center of origin
- disease progress curve
- epidemic
- epidemiology
- epiphytotic
- immunosuppressant
- infect
- infection period
- infest
- monoculture
- monocyclic
- mutagen
- mycotoxin
- polyculture
- polycyclic
- primary inoculum
- secondary inoculum
- severity value

Internet Resources

APS Resources

Bay laurel and native plants other than oaks are the main hosts for the sudden oak death pathogen, *P. ramorum*, in California
Citrus canker: The pathogen and its impact
Mycotoxins: The cost of achieving food security and food quality
Plant disease epidemiology (includes a simulation for different levels of primary inoculum and epidemic rate)
Revitalization of the majestic chestnut: Chestnut blight disease
What are the nonindigenous plant pathogens that threaten U.S. crops and forests?

Recommended Websites

California Oak Mortality Taskforce
Climate Change (Environmental Protection Agency)
Dogwood Anthracnose and Its Spread in the South
The Great Irish Famine Curriculum
North American Plant Protection Organization (NAPPO) Phytosanitary Alert System
ProMED Program for Monitoring Emerging Diseases, International Society for Infectious Diseases

Internet Research

Internet Research Exercise 10.1. Find news articles and/or scientific reports about a plant disease epidemic that has occurred within the past 5 years. How did people affect the course of this epidemic?

Internet Research Exercise 10.2. Go to the Phytosanitary Alert System of the North American Plant Protection Organization. This site includes links to other regional plant protection organizations (under the Resources link). Search for recent pest and pathogen problems on plants of interest to you.

Chapter 11

How Can We Prevent or Manage Plant Disease Epidemics?

IN THIS CHAPTER:

- ► How do we choose the appropriate methods for disease management?
- ► How do we **avoid** pathogens?
- ► How do we **exclude** pathogens?
- ► How do we **eradicate** pathogens?
- ► How do we **protect** plants?
- ► How can we create integrated, sustainable disease management programs?

How do we choose the appropriate methods for disease management?

Decades ago, this chapter would have discussed disease control, with the implication that plant diseases can be completely eliminated. Over the years, approaches to reducing plant diseases have evolved into forms of disease management. Unlike disease control, disease management attempts to maintain disease levels below **economic thresholds** because complete elimination of diseases is unnecessary and may result in unacceptable costs, labor, and environmental impacts. There are many approaches to the management of plant diseases. As the disease triangle reminds us, the appropriate choices depend on the pathogen, the plant, and the environment in which they interact. It also reminds us of the importance of accurate diagnosis in disease management. Using the disease pyramid, we also should remember that time can impact an epidemic. How long the pathogen, plant, and various environmental conditions interact will affect disease severity and, therefore, our management inputs.

As discussed in Chapter 10, most epidemics can be categorized as **monocyclic** or **polycyclic**. Although many epidemics fall in the spectrum between these two categories, they still help us to determine which management practices will be most useful. In general, management of a monocyclic epidemic should focus on reducing the amount or efficacy of the primary inoculum. Management of a polycyclic epidemic usually requires reducing the primary inoculum and also attempting to slow the rate of disease increase. The relative importance of these two approaches to polycyclic epidemic management depends on how fully the primary inoculum can be reduced or eliminated, the potential rate of disease increase, and how long the plants will remain susceptible to infection.

How do we avoid pathogens?

As discussed in Chapter 10, diseases have sometimes determined where crops are grown. Natural rubber originated in Brazil but is now grown mostly in Southeast Asia, because of South American leaf blight (Figure 11.1). Coffee originated in Ethiopia, but most coffee production is now in the Western Hemisphere because of coffee rust. In the United States, pears are mostly produced in drier regions instead of humid regions because of fire blight.

► DID YOU KNOW?

The rubber plant (*Hevea brasiliensis*) originated in Brazil. The first large plantation was established in Brazil at “Fordlandia” by the Ford Motor Company in 1927. By 1934, Ford had planted over 3,200 hectares (8,000 acres), but the rubber trees quickly began to die of South American leaf blight (SALB) caused by the polycyclic ascomycete *Microcyclus ulei*, which thrived in the wet tropical climate. Another 5,000-hectare (12,000-acre) plantation was established, but that too failed. Today, 90% of the world’s rubber comes from Southeast Asia to avoid SALB. The plants are protected by strict quarantines.

Figure 11.1. Rubber plantation in the Philippines.

Certain diseases can be reduced or managed through choice of the planting site or the planting date in a local area. The goal, to reduce exposure of the plant to inoculum, is essentially a form of escape. For example, damping-off often can be **avoided** by planting seeds in well-drained soils and at temperatures that minimize germination time. Seeds planted in cold, poorly drained soils are more likely to be attacked by damping-off pathogens. White pine blister rust hazard zones have been established in the western forests of the United States. By choosing upper slopes and ridges for planting rather than humid valleys, disease is minimized, even though not all of the wild *Ribes* alternate hosts have been eliminated. In California, growers honor a “lettuce-free” period for 2 weeks each year to break the cycle of aphid vectors and infection by *Lettuce mosaic virus* (Figure 11.2).

To Avoid Damping-Off in Outdoor Soils

- Prepare soil to have a uniform grade without low spots where water can accumulate.
- Avoid areas of soil compaction or poor drainage.
- Provide for good air movement across the planting area.
- Avoid planting seeds when excessive rainfall is expected.
- Choose a planting time when soil temperatures favor germination of seeds and air temperatures favor rapid growth and development of the plant.

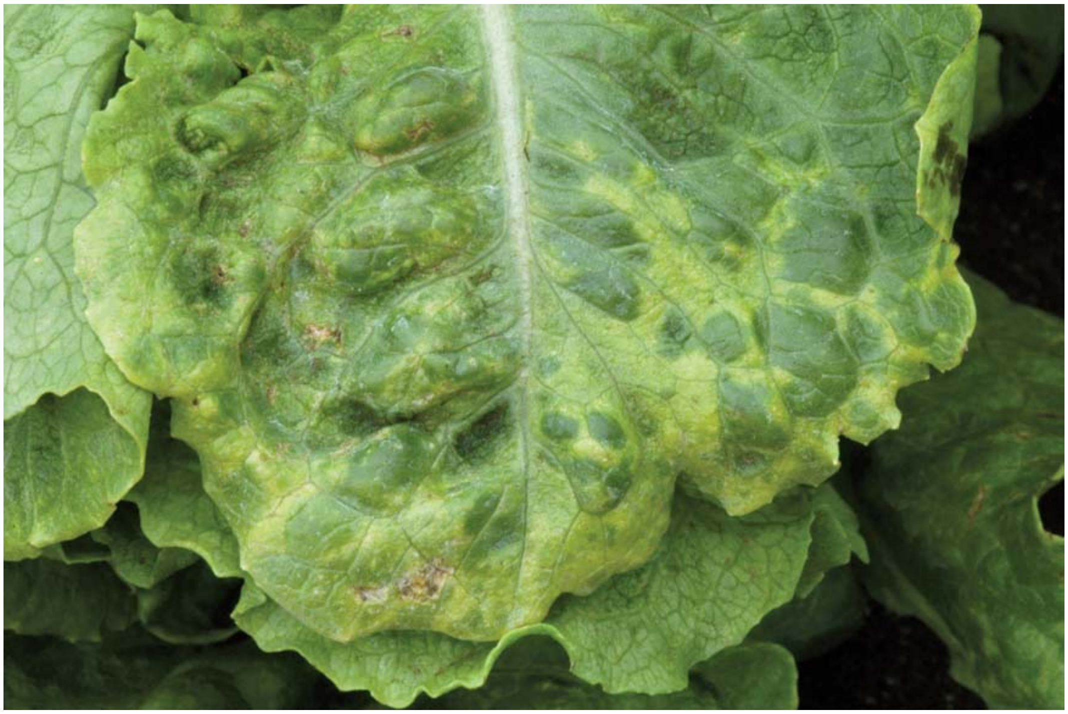


Figure 11.2. Romaine lettuce infected by *Lettuce mosaic virus*.

Home gardeners can try to avoid diseases when they establish their gardening areas. Open areas in full sun should be chosen for most flower and vegetable gardens. If soil is poorly drained, the construction of raised beds and the use of soil amendments will improve drainage and create a more favorable root environment. A soil sample should be sent to a local extension office or garden store to test for pH and soil nutrients. Sometimes lime or other amendments are necessary to optimize soil pH for plant growth and to avoid certain diseases. Fertilizers should be added as recommended by the soil test results. Finally, recommended planting times for seeds and transplants should be followed to encourage rapid establishment and to avoid damping-off pathogens and frost injury.

Unfortunately, it is not usually possible to completely avoid disease. Therefore, most disease management choices either reduce the amount or efficacy of inoculum (by exclusion and eradication) or protect the plant.

Avoidance focuses on the environmental component of the disease triangle. Pathogens can sometimes be avoided by the choice of the planting site or the timing of planting.

How do we exclude pathogens?

Exclusion (management of plant disease by preventing the introduction of pathogens to crop production areas) is usually considered to include various legal restrictions on movement of plants and pathogens, such as quarantines, and methods used to ensure that seed and other propagative parts are pathogen-free at planting time.

Methods of EXCLUSION

Quarantines: legislative control of the transport of plants or plant parts to prevent the spread of pests or pathogens

Pathogen-free seed: seed produced in areas with less disease; certification that seed meets established thresholds for exclusion of pathogens

Pathogen-free propagative parts: a combination of meristem-culture, heat treatment, and indexing for certain pathogens; certification that propagative parts meet established thresholds for exclusion of pathogens

Quarantines

From previous chapters, it should be clear that introduced pathogens have caused some of the most devastating plant diseases.

Plants and pathogens moved around the world without restriction for many years. The years 1800 to 1850 were a particularly active time for exploration to find and import interesting new horticultural and agronomic species.

The word ***quarantine*** is derived from the Latin word for 40, which refers to the number of days that passengers were held on ships after arrival in foreign ports to make sure that they were not bringing diseases to the cities. Apparently, anyone who was ill would have recovered or died by the end of that period. Today, the word *quarantine* relative to plant diseases refers to any restriction in the movement of plants, plant materials, seeds and propagative parts, soil, machinery, or anything that might introduce dangerous pathogens into new areas.

► **DID YOU KNOW?**

Phytophthora kernoviae is a recently discovered oomycete in the United Kingdom and New Zealand. Its hosts include beech, English oak, rhododendron, and wild *Vaccinium* spp. Depending on the host, it can cause leaf necrosis, stem dieback, and "bleeding cankers." It is considered a more destructive pathogen of rhododendrons than *P. ramorum*, the causal agent of sudden oak death in California. In 2005, the U.S. Department of Agriculture notified the European Union that nurseries wishing to export host plants to the United States must have an annual inspection to be sure that the nursery is free of *P. kernoviae*.

The first U.S. plant quarantine law was passed in 1912 following the introduction of citrus canker, chestnut blight, and white pine blister rust. These diseases had devastating impacts on their host plants and caught the attention of legislators. Many other important pathogens of fruit, vegetable, and field crops have been introduced into the United States, and many have been spread from the United States to other parts of the world over the years.

Tourists are not allowed to bring any fresh plant materials, including flowers, fruits, seeds, and vegetables, into the United States (Figure 11.3). These materials are confiscated because it is not usually possible to identify the presence of plant pathogens or early infections from cursory visual inspections. Around the world, travelers may be questioned about visits to farms because boots may carry infested mud, or they may even find their airplane cabin sprayed with an insecticide before they are allowed to disembark from the plane.

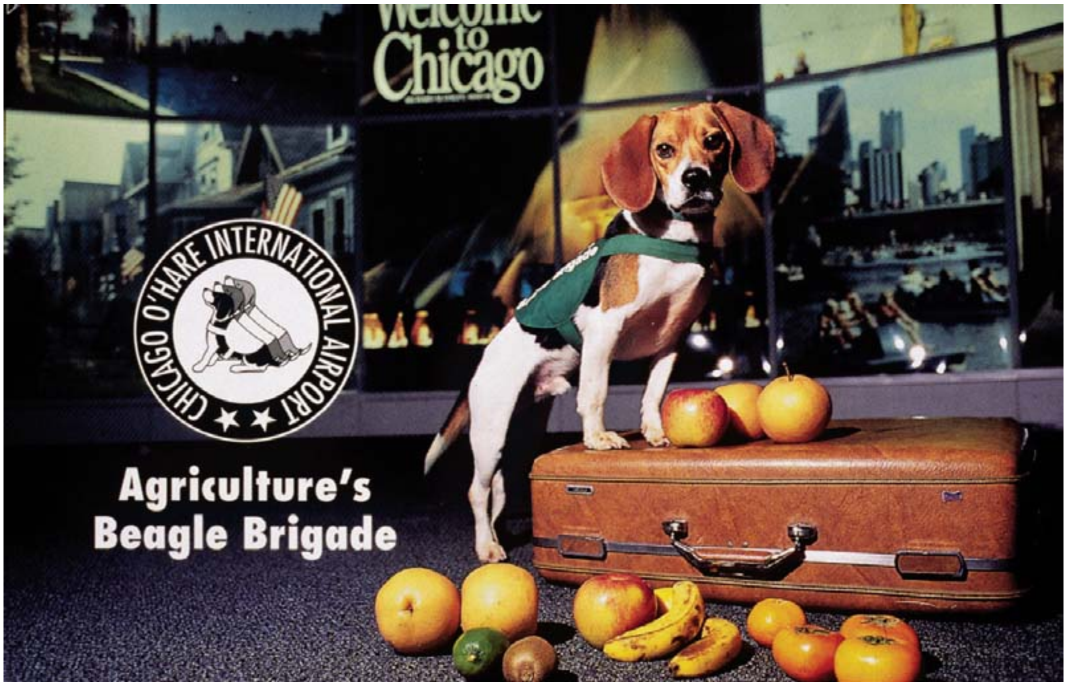

Welcome to Chicago
CHICAGO O'HARE INTERNATIONAL AIRPORT
Agriculture's Beagle Brigade

Figure 11.3. Beagles are trained to help detect illegal fruits and vegetables at ports of entry.

Commercial plant producers are subject to different restrictions and must apply for permits to import plant materials. Some items are **unrestricted**, but most fall into the **restricted** category, which requires inspection, and sometimes chemical treatment, before release. The **post-entry** category requires that plants be kept in isolation for up to two years and be subject to additional inspections during that time since pathogens may not be obvious at the time of import. A few kinds of plant materials from certain areas are considered so dangerous that they are completely **prohibited**. Imported plant materials, including seed, must receive a **phytosanitary certificate** (see Figure 4.17) before they can be imported or exported across national boundaries, according to the regulations of the importing country.

Import of Plant Materials into the United States

Tourists

- No plant materials may be imported by tourists.

Commercial plant material import categories

- unrestricted
- restricted
- post-entry (must be maintained for continued inspection for up to two years)
- prohibited

Local quarantines are sometimes established by regional areas, such as states. For example, a number of agricultural pests and pathogens are present in Hawaii but not on the mainland of the United States and vice versa, so quarantine inspections may be required. In 2006, quarantines were in effect for citrus canker in Florida; the golden nematode on Long Island, New York; *Plum pox virus* in Pennsylvania; and witchweed in North and South Carolina. In addition to these official quarantines, seedlings and other plants may be subject to inspections before interstate shipment. A website at the end of this chapter lists the pests and pathogens that are under quarantine for various U.S. states.

One of the major limitations of quarantines is that they are based on political boundaries that may or may not correspond to biological boundaries. Quarantines are not useful where natural dispersal by air, vectors, or water is likely to occur. For example, most quarantines between the United States and Canada are for soilborne pathogens rather than for airborne pathogens. Airborne pathogens can sometimes move quickly over large areas. For example, in a single year (1978), the sugarcane rust fungus was naturally distributed from Africa to the Dominican Republic and on to Florida in the United States.

Limitations to Successful Quarantines

- natural dispersal of pathogen or vector
- inability to see microscopic pathogens or early infections
- language differences among countries
- insufficient resources and/or technical training
- need for rapid testing and risk assessment to meet commercial import and export needs

Another limitation to successful quarantines is the inability to easily determine whether plant materials carry pathogens. Many countries do not have sufficient resources or trained inspectors. Language differences and variable regulations impede successful quarantines, especially for land-locked countries that share their borders with several different countries. Quarantine enforcement for the United States, bounded only by Canada, Mexico, and two oceans, is less challenging in comparison.

The globalization of trade is an important threat to the successful enforcement of quarantines. Inspectors must quickly decide whether large shipments of fruits, vegetables, cereal crops, and wood products are carrying dangerous pests, pathogens, or vectors. Rapid detection methods such as DNA-based tests (e.g., PCR) and immunoassays have improved our ability to test materials quickly and accurately, but these methods are expensive and require that the inspectors know what pathogens might be present (see Appendix 3).

> “One of the unintended consequences of the globalization of trade has been an increase in the frequency of introductions and the number of exotic (alien, introduced, foreign, non-native, or non-indigenous) species intercepted at ports of entry.”
>
> —L. D. Dwinnel, 2001, “Exotic Forest Pests—A Global Issue”

To remain competitive, growers who produce flowers, vegetables, fruits, and ornamentals must meet market demands, which means that they routinely import new cultivars. These growers want the new plant materials released for propagation as quickly as possible. The employees of the U.S. Animal and Plant Health Inspection Service (APHIS), state diagnostic laboratories, and comparable government agencies in other countries are under increasing pressure to monitor more (and different) plants and plant products every year. They must quickly conduct risk assessments for numerous pathogens, asking such questions as:

Is the pathogen capable of causing serious damage?

Is introduction by natural dispersal unlikely?

Is the pathogen capable of becoming established anywhere in the country, including greenhouses?

In most cases, quarantines are only a delaying mechanism, which give plant pathologists time to devise management programs and develop resistant cultivars.

Quarantine Legislation and Organizations

Plant Quarantine Act of 1912: first U.S. legislation establishing plant quarantines

Animal and Plant Health Inspection Service (APHIS): federal agency responsible for quarantines

Plant Protection and Quarantine (PPQ): branch of APHIS that issues federal phytosanitary certificates for exporters

International Plant Protection Convention (IPPC): sponsored by the Food and Agriculture Organization (FAO) of the United Nations in 1952 to provide member countries with phytosanitary export permits for eight regional organizations, including

- **European and Mediterranean Plant Protection Organization (EPPO)**
- **Intraafrican Phytosanitary Council (IAPSC)**
- **North American Plant Protection Organization (NAPPO)**

► DID YOU KNOW?

Black knot disease of plum and cherry is mainly a problem in North America, where it is indigenous. It is not yet found in European or Mediterranean countries. Currently, its pathogen, *Apiosporina morbosa*, is listed as an EPPO quarantine pathogen.

Plant Disease Profiles: Black knot

Pathogen-Free Seed

Seedborne diseases are of particular concern because they introduce the pathogen along with the plant and can cause devastating losses (Figures 11.4 and 11.5). People were aware of this problem even before they understood its nature. For example, they soaked wheat and other cereal grains in copper, urine, and various concoctions to try to reduce problems with smut diseases. Some of these early investigations added evidence that led to the eventual acceptance of the germ theory of disease.

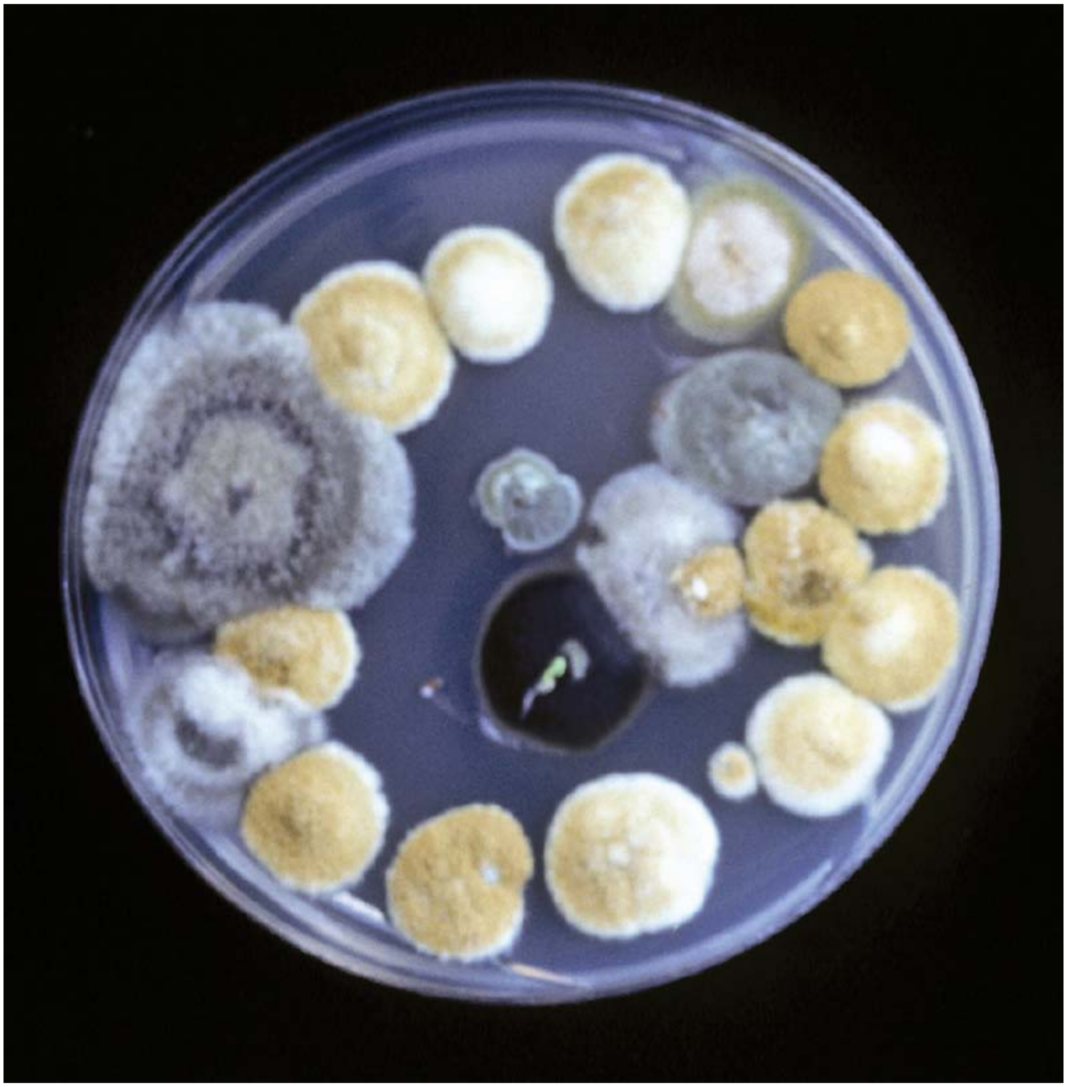


Figure 11.4. A variety of fungal contaminants growing from infested seed placed on a nutrient medium.

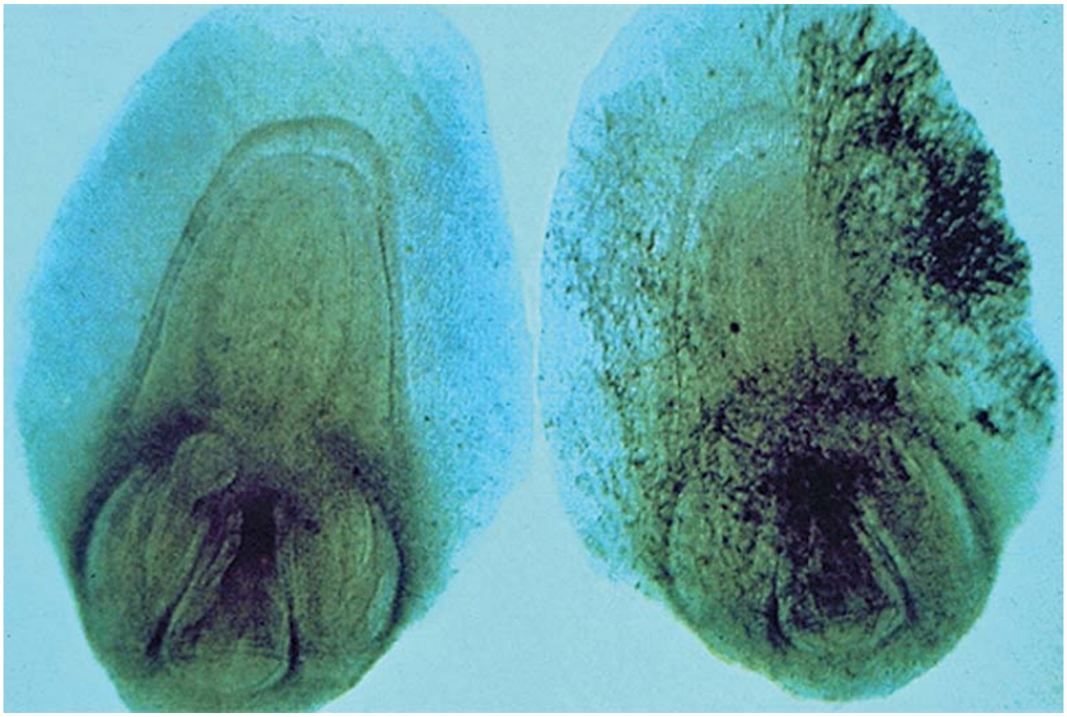


Figure 11.5. Barley seed embryo infected by the loose smut pathogen (*Ustilago nuda*) (right) and an uninfected embryo (left).

In the United States, seed production of many flower and vegetable crops is now centered in the drier western states, where plants can be grown in the near absence of seed-borne fungal and bacterial pathogens. By starting with this pathogen-free seed, susceptible crops can be grown in humid climates.

Seedborne Pathogens

	Infested seed	Infected seed
fungi	×	×
bacteria	×	×
viruses	uncommon	×
nematodes	uncommon	uncommon

Certification schemes determine what percentage of infested or infected seed would still allow a viable crop to be grown. Seed lots are then tested and certified as meeting these standards before seeds are sold. Certification is particularly important for many seedborne viruses because no effective seed treatments exist. Acceptable levels of seedborne pathogens vary, depending on the needs of the growers. In the United States, the threshold for *Lettuce mosaic virus* is 0 infected seeds in 30,000 tested, whereas in the Netherlands, it is 0 in 2,000 because disease pressure is lower there.

To Produce Certified Pathogen-Free Seed

- Grow mother plants in dry regions when possible.
- Isolate seed-production fields from commercial fields that may be a source of pathogens and/or vectors.
- Use careful water management to minimize leaf wetness.
- Remove infected or off-type plants during production.
- Test seed lots as required to meet certification standards for pathogens.

► **DID YOU KNOW?**

In 2009, three agencies of the U.S. Department of Agriculture established a National Clean Plant Network (NCPN). The participating agencies are the Animal and Plant Health Inspection Service (APHIS), Agricultural Research Service (ARS), and Cooperative State Research, Education and Extension Service (CSREES). The goals of the NCPN are to support research to produce pathogen-free plants and germplasm collections, to identify plant diseases, to develop methods to treat diseased plants, and to provide outreach and education in support of these efforts.

Pathogen-Free Vegetative Propagation

Propagative parts such as bulbs, corms, and tubers are likely to carry pathogens in their tissues or in soil attached to their outer layers. Modern plant production uses vegetative propagation for rapid multiplication of many plants, including fruit trees, flower crops, ornamentals, small fruits, potato, and sweet potato. The mother plant used to propagate numerous new plants should be free of systemic pathogens before propagation begins.

A method of culture **indexing** began in the flower industry to test mother plants for pathogens (see box “Meristem Culture and Certification for Vegetative Propagation”). In the early days, stem samples were placed in liquid media to look for bacterial growth or on solid nutrient media to test for fungal infection; various bioassays were used to test for viruses. Plants that tested clean would then be maintained under very strictly controlled conditions to prevent new infections. A small **mother block** of apparently pathogen-free plants was used to create **foundation** stock. These plants were generally still too expensive to use for production, so propagation from the foundation stock created various categories of **certified** stock that were eventually sold to producers.

Meristem Culture and Certification for Vegetative Propagation

Heat treatment (30–40°C, or 88–104°F) of plant (for 6–12 weeks)

↓

Excise meristem tip and culture on defined medium.

↓

Regenerate plantlet by manipulation of media.

↓

Establish plantlet in soil.

↓

Index for pathogens using:

- indicator plants
- culture media for fungi and bacteria
- grafting
- immunoassays and DNA-based assays

↓

Mother block or nuclear block of pathogen-free plants
maintained in restricted area with frequent retesting for pathogens

↓

Foundation plants
generated from mother block

↓

Certified plants
various levels, defined by number of years away from Foundation stock; must meet certification standards for pathogens

↓

Production stock

Although this was an effective way to reduce a number of systemic pathogens, it was not cost effective for most kinds of vegetatively propagated plants. However, a new technology was developing at about the same time that indexing was created. As scientists became familiar with plant growth regulators, they discovered that tiny pieces of tissue including the apical meristem could be cut and used to propagate almost unlimited numbers of new plants.

Not only were the meristems generally free of pathogens, but it was economically feasible to propagate large numbers of pathogen-free plants using **meristem culture** (see Figures 5.13 and 5.14). The tiny plants are grown in pathogen-free laboratories on sterile growth media containing various plant growth regulators until they are ready to be transferred to a greenhouse (Figure 11.6). In some cases, plants are sent to production fields directly from the greenhouse. More often, they are grown for a year or more outdoors for additional propagation before they are finally used in commercial production. These plants must meet various established certification requirements; limits are set for pathogens that are likely to cause severe disease problems that usually cannot be managed by means other than exclusion.

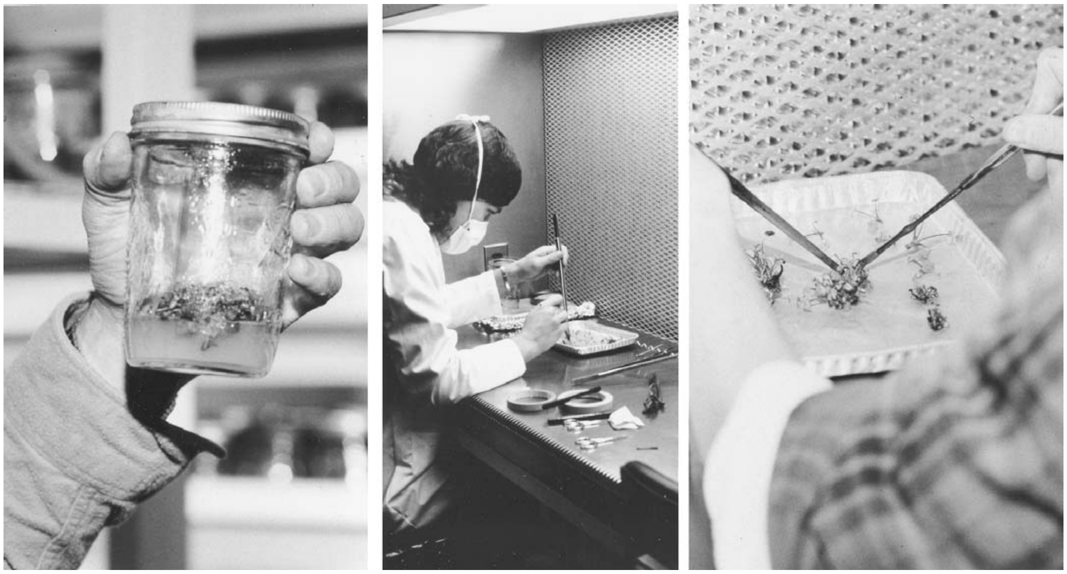


Figure 11.6. Regeneration of plants from a meristem. Left, Growth of the meristem tissue on a nutrient medium containing appropriate growth regulators to produce multiple shoots. Center and right, Under sterile conditions, the tiny shoots can be separated and grown into plants.

Exclusion of the pathogen is useful for both monocyclic and polycyclic pathogens. When successful, no disease will occur. Some pathogens can be excluded, at least temporarily, by quarantines. Regulations must accommodate the import and export needs of international trade. The production of pathogen-free seed and propagative parts depends on research-based certification standards and the ability to produce affordable planting material. Indexing tests plants for a variety of systemic pathogens. Meristem culture revolutionized the production of affordable pathogen-free plants.

How do we eradicate pathogens?

It is quite common for new cultivars or other desirable plants to be infected by pathogens. In addition, soil, tools, and machinery may be infested with pathogens. Therefore, it often is necessary to **eradicate** (remove or destroy) pathogens before production of healthy plants can occur.

Eradication can be organized under a series of approaches, all of which attempt to reduce the amount or efficacy of primary inoculum. Eradication implies complete elimination, which is not usually feasible or necessary for endemic pathogens. Furthermore, one needs to consider the cost-benefit relationship since complete eradication is much more expensive than partial eradication of inoculum. Methods for eradication include a range of cultural practices, chemicals, and biological controls. The following methods are commonly used to eradicate pathogens.

Methods of ERADICATION

Cultural practices

- crop rotation
- fallowing
- flooding
- host eradication
  - following failed quarantines
  - roguing of infected plants
  - destroying alternate or alternative hosts
  - plowing or tillage
  - sanitation

Heat

- burning
- steaming of soil, pots
- composting
- solarization
- hot-water treatments
- thermotherapy

Chemicals

- disinfestants
- herbicides for pathogen reservoirs
- insecticides for vectors
- seed treatments
- soil treatments (fumigants or nematicides)

Biological control

- hyperparasitism
- predation
- hypovirulence

Cultural Practices

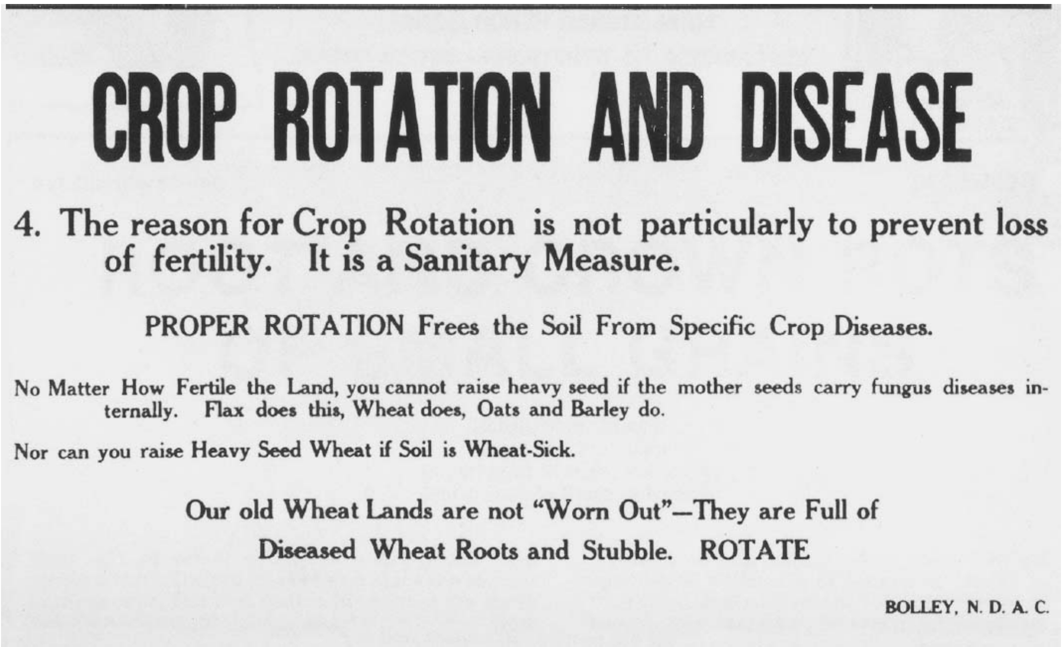
Many of the cultural practices used to eradicate pathogens are done before planting and are most effective for annual crops. In some cases, living host plants near the crop are removed because they are a source of inoculum. In other cases, practices such as tillage, crop rotation, and flooding help to reduce surviving populations of pathogens from previous crops. Sanitation may be done at the end of the growing season by cleaning up pathogen-infested debris or during the growing season by removing ("roguing out") infected plants or plant parts. Sanitation of irrigation water is important when it is shown to be a significant source of pathogens or is being recirculated.

CROP ROTATION

Rotation to a nonhost crop (Figure 11.7) forces pathogens to persist as survival structures and/or as saprophytes. Starvation of the pathogen is a key mechanism of crop rotation. There are several important economic or biological limitations to the use of crop rotation. Some pathogens are good soil inhabitants, so their populations do not decline rapidly even in the absence of a host plant. Other pathogens have wide host ranges, limiting the choices for nonhost crops and allowing survival on weed species or on host species in nearby natural areas. Rotation is not an option for perennial agricultural systems such as golf courses, forest crops, nurseries, and small fruits. Specialized equipment used for planting and harvesting may limit crop choices available to a grower. Finally, crops vary in their value, so there is sometimes an economic incentive to produce a high value crop as often as possible.

Limitations to the Use of Crop Rotation

- perennial crops
- specialized equipment required for producing and harvesting some crops
- economic value of alternative crops may be too low
- long-lived pathogens in soil (soil inhabitants)
- pathogens with a wide host range
- pathogens with efficient methods of long distance dispersal



CROP ROTATION AND DISEASE

4. The reason for Crop Rotation is not particularly to prevent loss of fertility. It is a Sanitary Measure.

PROPER ROTATION Frees the Soil From Specific Crop Diseases.

No Matter How Fertile the Land, you cannot raise heavy seed if the mother seeds carry fungus diseases internally. Flax does this, Wheat does, Oats and Barley do.

Nor can you raise Heavy Seed Wheat if Soil is Wheat-Sick.

Our old Wheat Lands are not "Worn Out"—They are Full of Diseased Wheat Roots and Stubble. ROTATE

BOLLEY, N. D. A. C.

Figure 11.7. A poster by H. L. Bolley, from North Dakota in 1909, explaining that the benefits of rotation of wheat with other crops result from a reduction of soilborne pathogens.

FALLOWING

Leaving a field free of a crop as part of a rotation plan has been practiced historically and is practiced today in subsistence farming and dry-land agriculture. Fallowing is not an effective strategy for pathogens that can survive on weed hosts or for long periods of time in the soil. In many areas, fallowing is no longer economically feasible because a food, forage, fiber, or cash crop must be obtained from all fields each year.

FLOODING

Flooding soil creates anaerobic conditions that reduce pathogen populations. This ancient agricultural method is widely used in rice culture (Figure 11.8). It also is used before planting to reduce Panama wilt (Fusarium wilt) in banana and for nematode problems in certain crops.

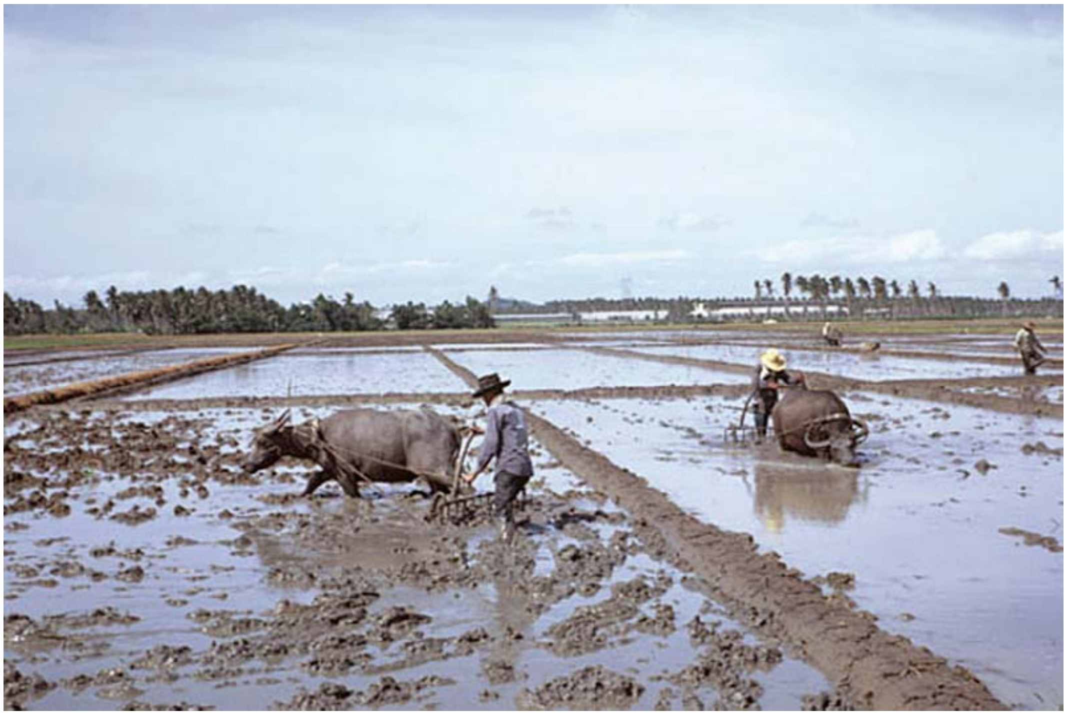


Figure 11.8. Flooding and tillage before rice cultivation in the Philippines.

HOST ERADICATION

There are several situations in which infected plants may be eradicated to remove sources of primary inoculum. For example, quarantines sometimes fail despite our best efforts. The first step to control an introduced pathogen is to try to eradicate the infected plants before the pathogen can spread, although that is not always successful. Citrus canker became a problem again in Florida in 1984 despite its apparent eradication decades earlier in the citrus-growing states of the United States. Quarantines followed in an attempt to prevent its spread, but due to three hurricanes and a tropical storm in 2004, these ultimately failed. Quarantine activities within Florida have now been converted into management programs, although quarantines remain in effect for other citrus-growing areas of the United States. When, despite quarantines, *Plum pox virus* (Figure 11.9) was discovered in orchards in Ontario, Canada, and in Pennsylvania in the 1990s, a buffer of 20 stone fruit trees was cut down around every infected tree, and roots were pulled out to prevent regrowth. Coffee rust was found several times in South America in the 1970s, and attempts were made to eradicate it. However, it is now well established in South America and most of the other coffee-growing areas of the world.

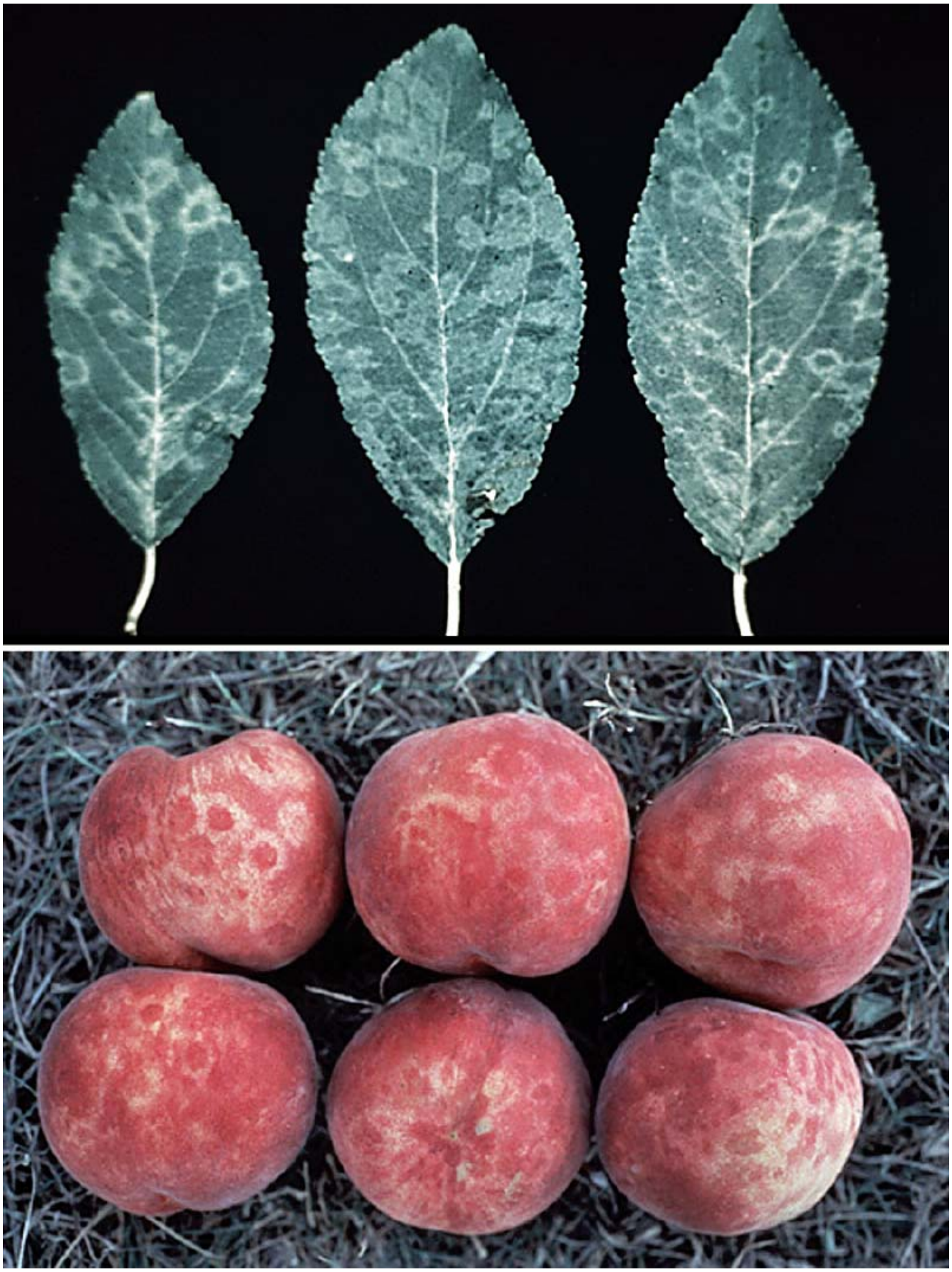


Figure 11.9. Symptoms of *Plum pox virus* of stone fruits.

Volunteer plants are those that grow from leftover seeds or propagative parts of the previous crop. They are sometimes sources of primary inoculum for the newly planted crop and should be removed. When current-season crop plants first emerge, it is a common practice to visually inspect them for symptoms of disease. Infected individuals are removed, so that they do not serve as a source of inoculum for neighboring plants, a practice called **roguing**. For example, certification of potato plants grown for seed-potato production requires that the fields be inspected for virus symptoms and certain other diseases on several occasions during the growing season (Figure 11.10).

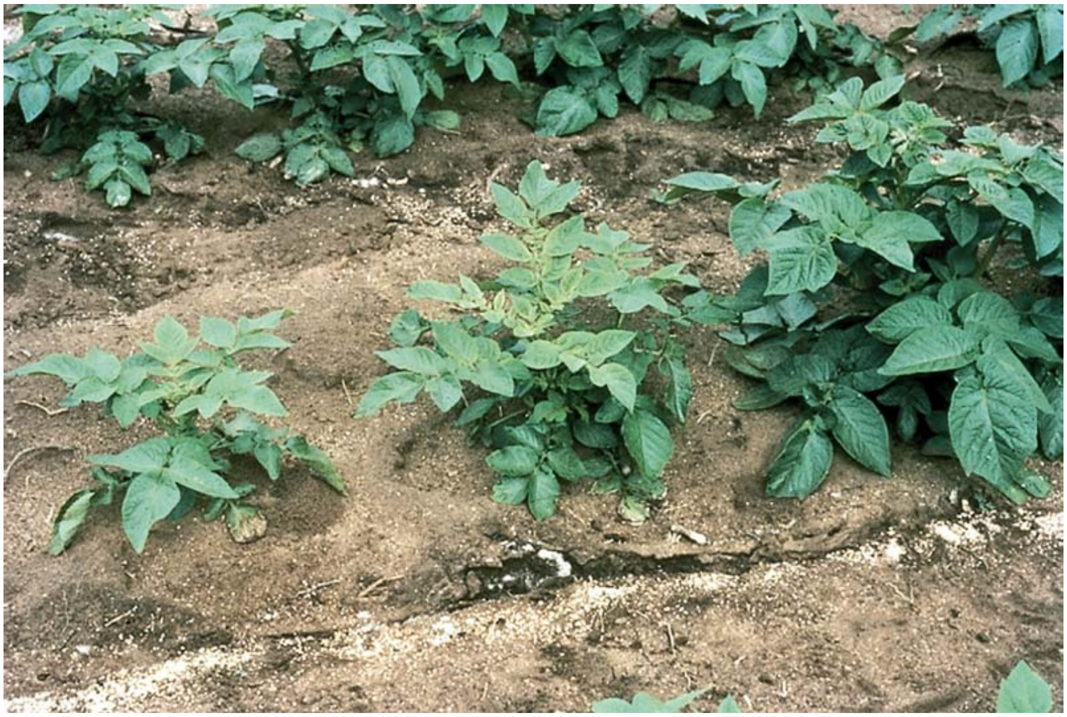


Figure 11.10. Virus-infected potato plants should be removed to prevent the spread of the virus to healthy plants.

Alternate hosts of rust fungi can be eradicated to break the infection cycle. The barberry and *Ribes* eradication campaigns attempted to protect wheat from stem rust and white pines from blister rust, respectively. Junipers (red-cedars) often are eradicated near apple orchards to reduce cedar-apple rust. It also is common to try to remove **weeds** and other **alternative hosts** that are sources of inoculum of viruses, fastidious bacteria, and some nematodes. For example, wild cherry trees near peach and cherry orchards can serve as sources of X disease phytoplasma inoculum and should be eradicated.

► **DID YOU KNOW?**

Laws banning the planting of barberry near wheat fields were first passed in Rouen, France, in 1660, almost 200 years before the germ theory and an understanding of the heteroecious nature of *Puccinia graminis*. European colonists brought barberry, along with wheat, to North America. The thorny barberry bushes were used for hedges to contain animals; the wood made good tool handles; and the bark produced a yellow dye. Barberry laws were enacted in several New England colonies in the mid-1700s; however, barberry continued to spread as pioneer farmers moved west. More than 295 million barberry bushes and seedlings were killed during the barberry eradication campaign of 1918–1941. School children who participated received "Rust Buster" certificates.

PLOWING (TILLAGE)

As discussed previously, plowing stubble and debris from previous crops into the moist soil environment speeds decay of plant debris and forces soil invaders into competition with soil inhabitants more quickly.

SANITATION

A variety of basic sanitary practices can eradicate pathogens. Cull piles of infected plant materials should not be left just outside greenhouses or at the edges of fields after harvest, where spores or pathogen-carrying vectors can easily travel to new healthy plantings

(Figure 11.11). Cleaning up infested debris or fallen leaves is sometimes practical and effective. Infested tools, machinery, and boots should be cleaned before being moved to areas where a pathogen is not active or established (Figure 11.12). Sometimes a simple water rinse is sufficient, but in other cases disinfestants are applied to machinery, tools, and tires. Infected plant parts can sometimes be pruned away in the early stages of infection, especially in woody plants.

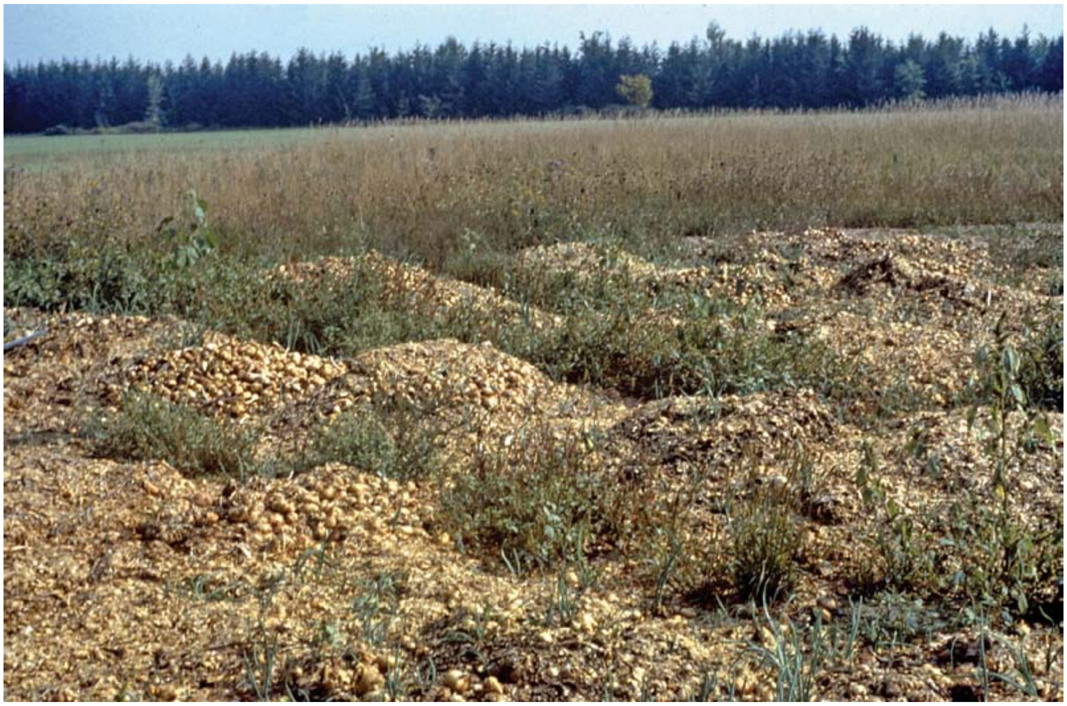


Figure 11.11. Onion cull piles are a source of *Onion yellow dwarf virus*, which is spread by aphid vectors.

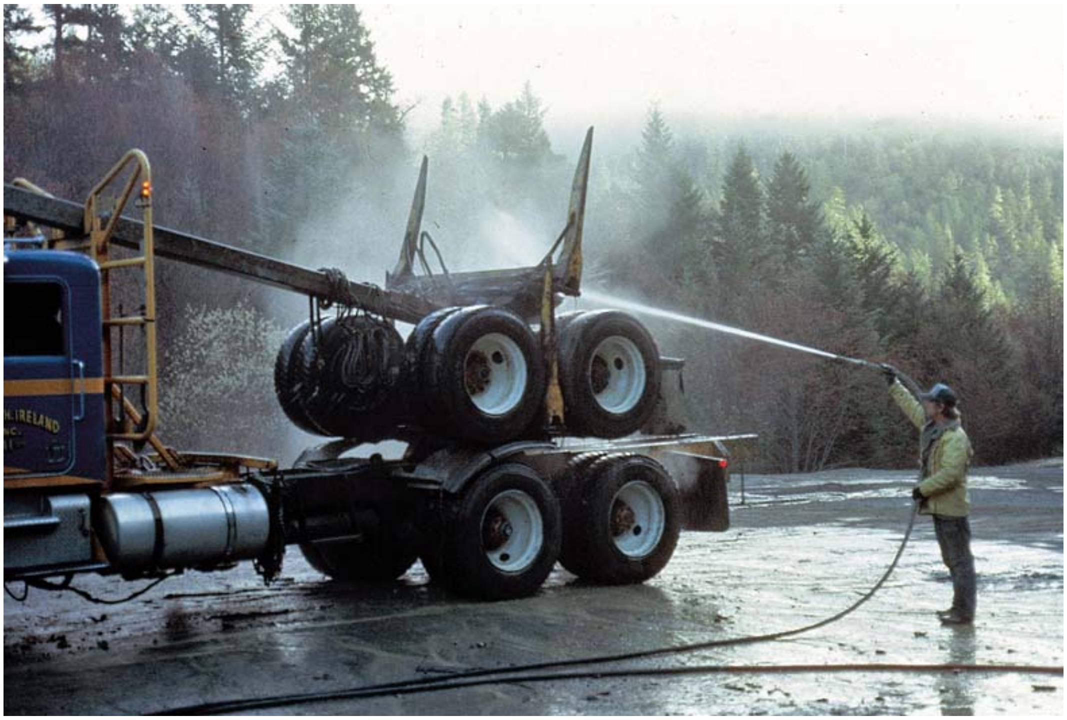


Figure 11.12. Washing a log truck before it enters a "clean" area to prevent the spread of *Phytophthora*, a soilborne pathogen.

Water is an important source of many pathogens, including bacteria, nematodes, *Fusarium* spp. and other fungi, oomycetes such as *Pythium* and *Phytophthora* spp., and viruses. Pathogens may be found in irrigation water, leachate from pots, "ebb and flow" irrigation systems on greenhouse benches, and water in hydroponic systems. Irrigation ponds are the primary sources of *Phytophthora* diseases in fruit, nursery, and vegetable crops. As water conservation, reuse, and recirculation become more important, a variety of sanitary practices may be employed to reduce pathogens, including filtration and treatment with chlorine, ozone, ultraviolet radiation, or heat. Sanitary practices also must be used to reduce potential water contamination from sources of infested plant residues or pathogen-contaminated soil, such as shoes, tools, or run-off from cultivated areas.

Heat

As can be seen in Figure 11.13, relatively low temperatures can effectively eliminate many pathogens.

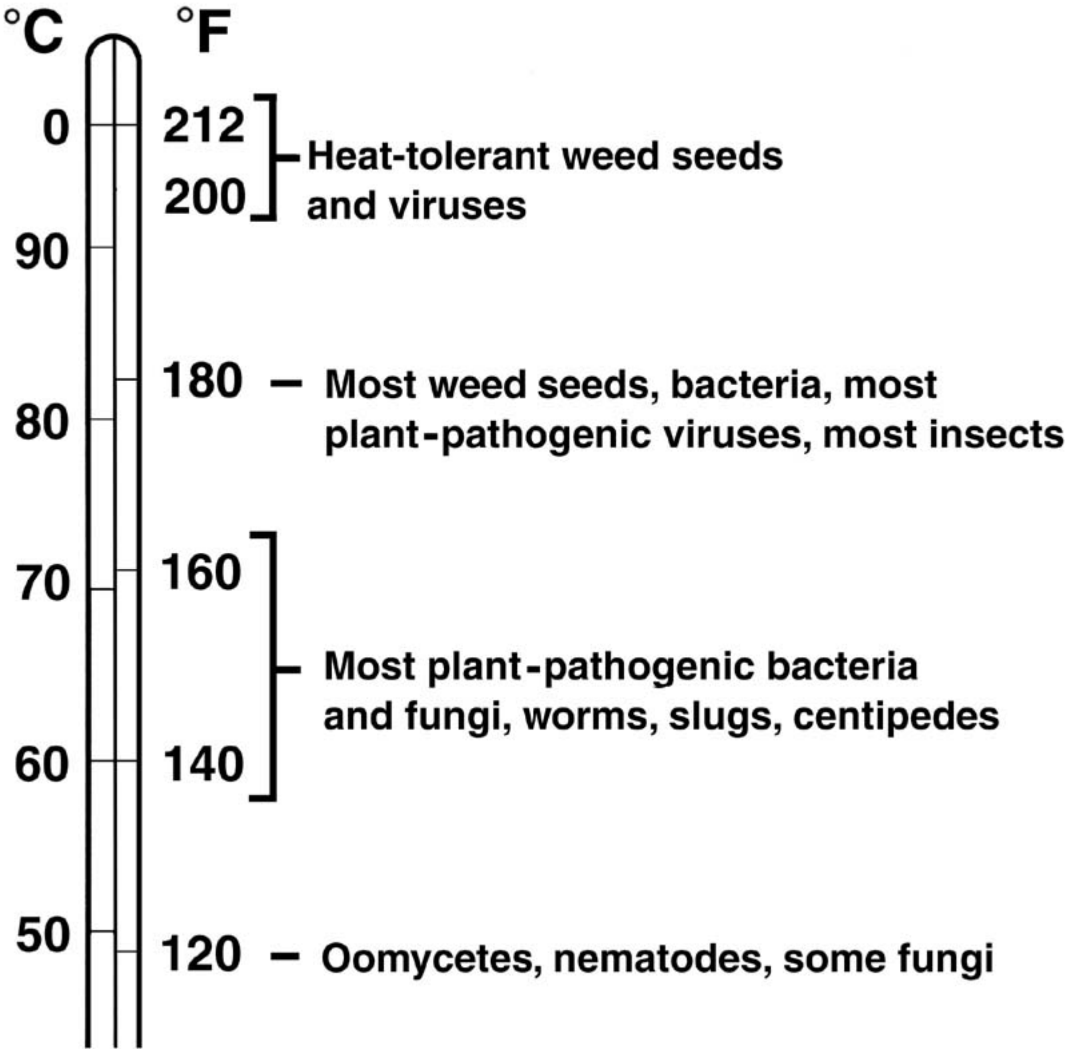

°C
°F
0
212
200
Heat-tolerant weed seeds and viruses
90
180
Most weed seeds, bacteria, most plant-pathogenic viruses, most insects
80
70
160
Most plant-pathogenic bacteria and fungi, worms, slugs, centipedes
60
140
50
120
Oomycetes, nematodes, some fungi

Figure 11.13. Temperatures necessary to kill pathogens and other plant pests. Most of the temperatures are for a 30-minute exposure under moist conditions.

BURNING OF STUBBLE AND DEBRIS

This ancient practice reduces weeds, pests, and pathogens, especially those near the soil surface. These benefits also probably accompany traditional “slash and burn” agriculture. Grass seed production fields, peppermint fields, and hops yards all are burned using techniques that reduce pests and pathogens but do not kill these perennial plants (Figure 11.14). Infested debris that has been pruned or removed from fields is sometimes burned to prevent inoculum from spreading to nearby plants (Figure 11.15).

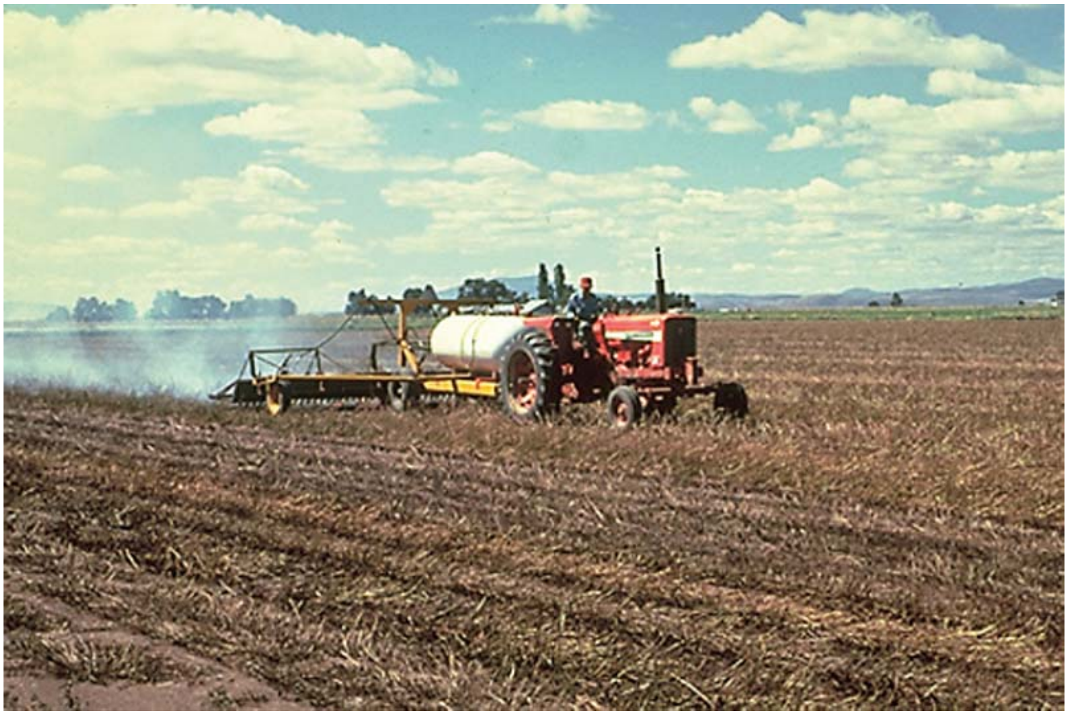


Figure 11.14. Propane flaming of peppermint debris after harvest reduces *Verticillium* inoculum entering the soil.

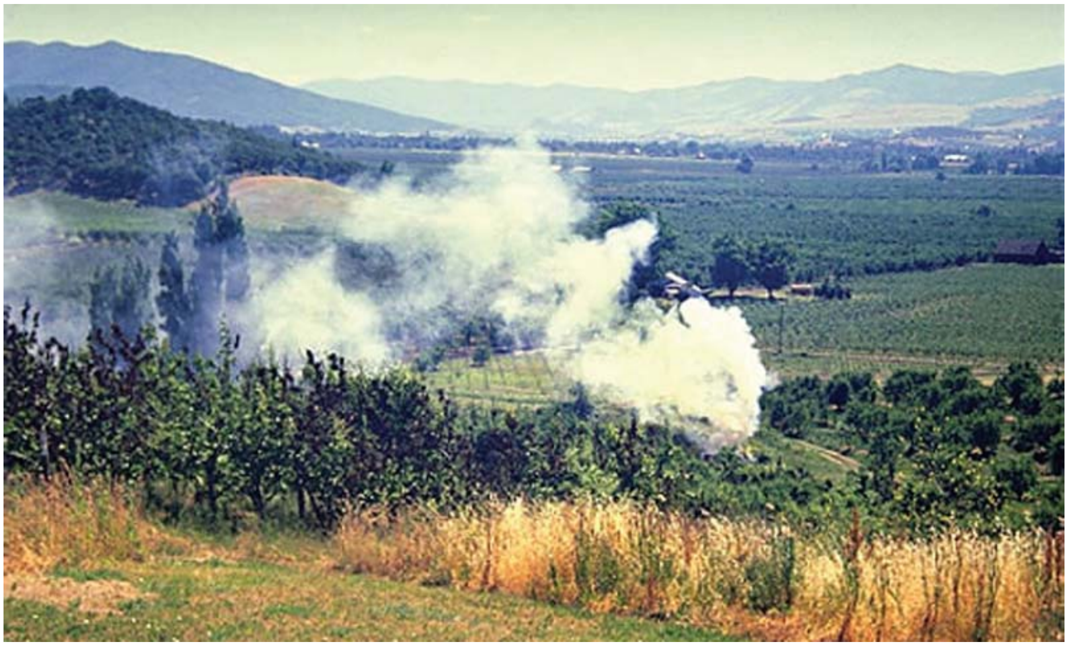


Figure 11.15. Burning of fire blight prunings in a pear orchard.

STEAM HEAT

Steam heat is commonly used to disinfest pots, soil, and other planting media used for potted plants in nurseries and greenhouses. Soil-free planting media usually have fewer pathogens than native soils, but it is easy for these products to become contaminated with pathogens, so they are usually pasteurized before use. Many purchased potting mixes have already been heat-treated before packaging. As discussed in Chapter 9, soil pasteurization is designed to heat soil to 60–70°C (140–160°F) to kill most pathogens but not the protective soil inhabitants. This also helps to retain good soil structure compared to soil sterilization with live steam. In some cases, steam heat is used for outdoor soils, but the time required, the inconvenience, and the expense usually limit this to flower beds or other small areas.

COMPOSTING

This is an effective method to reduce pathogens. Composted organic materials provide a favorable environment for the growth of thermophilic (high-temperature-loving) organisms that sufficiently compete with the pathogens, which are generally adapted to ambient conditions. Effective composting requires adequate heating and frequent turning of the pile to expose all of the materials to moisture and microbial activity. Composted plant debris is a good source of nutrients and organic matter for soil amendments, but it is usually recommended that plant debris that is heavily infested with pathogens should be buried or destroyed rather than used for compost.

SOLARIZATION

The process of trapping solar radiation under clear plastic sheets to heat the soil and reduce pathogens, pests, and weeds is called **solarization** (Figure 11.16). This can be an effective way to reduce primary inoculum because many important pathogens are killed at relatively low temperatures. It is most effective for pathogens that are near the soil surface, but it does not control some heat-tolerant pathogens such as root-knot nematodes. A significant amount of disease reduction may be the result of increased activity of competing and antagonistic organisms rather than of the heat killing the pathogen directly. Solarization usually should be done during the hottest part of the year, so it can be used during the summer in greenhouses that are used for plant production during the cooler months. A limitation to the use of solarization is the relatively long time (6–8 weeks) that the procedure requires, which may result in taking crop acreage out of production during all or part of the growing season. Additional limitations are the need for access to uninterrupted intense solar radiation, the storage or disposal of large amounts of plastic, and the cost.

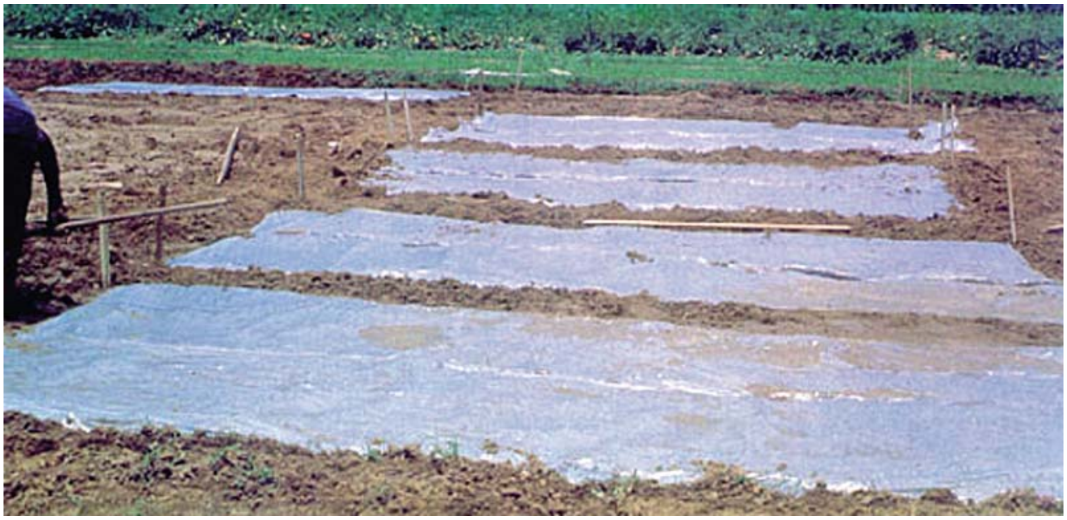


Figure 11.16. Soil solarization. Soil is treated by covering it with clear plastic sheeting for 4–8 weeks.

HOT-WATER TREATMENTS

Hot water is sometimes used to kill pathogens in seeds and propagative parts. Of course, one must take care not to kill embryos or buds in the process of killing the pathogen. Hot water treatments for seeds have been used for bacterial and fungal pathogens. The production of seed in drier climates has greatly reduced the need for seed treatment for bacteria, and systemic fungicides have greatly reduced the use of hot water seed treatment for fungi. Hot water dips are occasionally used for nematodes such as *Ditylenchus* spp. in bulbs.

THERMOTHERAPY OF PLANTS OR PLANT PARTS

To eradicate viruses before propagation, long periods (6–10 weeks) of heat treatment (30–40°C/86–104°F) have been used to obtain virus-free meristems from virus-infected plants. Heat treatment has become a routine step in meristem culture, even for mother plants that have tested virus-free, because unknown systemic pathogens might be present. Thermotherapy is thought to be effective because the high temperatures reduce virus replication, and plasmodesmata (cytoplasmic strands or channels through which viruses move from cell to cell) are not developed in meristems.

Heat is a very effective means of eradicating pathogens. High temperatures can be used to disinfest soil, tools, and pots. Lower temperatures must be used on living plant materials such as mother plants for meristem culture, propagative parts, and seeds, but, fortunately, many pathogens are quite sensitive to even moderate heat.

Chemicals

Various chemicals have been used in plant disease management. In some cases, they are used to eradicate the pathogen from soil, tools, machinery, seeds, or propagative parts. Other chemicals are applied to growing plants to protect them from infection or to eradicate very early infections. The use of chemicals as plant protectants is discussed in the Protection section of this chapter.

Generic Terms for Pesticides

- antibiotics (bacteria)
- biocides (broadly toxic—used for some soil fumigants)
- fungicides (fungi)
- herbicides (plants)
- insecticides (insects)
- nematicides (nematodes)
- rodenticides (rodents)

DISINFESTANTS

Alcohol, bleach (sodium hypochlorite), and quaternary ammonium are common disinfestants. They are used to clean storage areas for harvested fruits, vegetables, seed, and propagative parts for the next planting, as well as boots, greenhouse benches, tools, machinery, and vehicles (Figure 11.17). The concentration of the disinfestant and the exposure time needed to eradicate pathogens vary depending on the material to be disinfested.

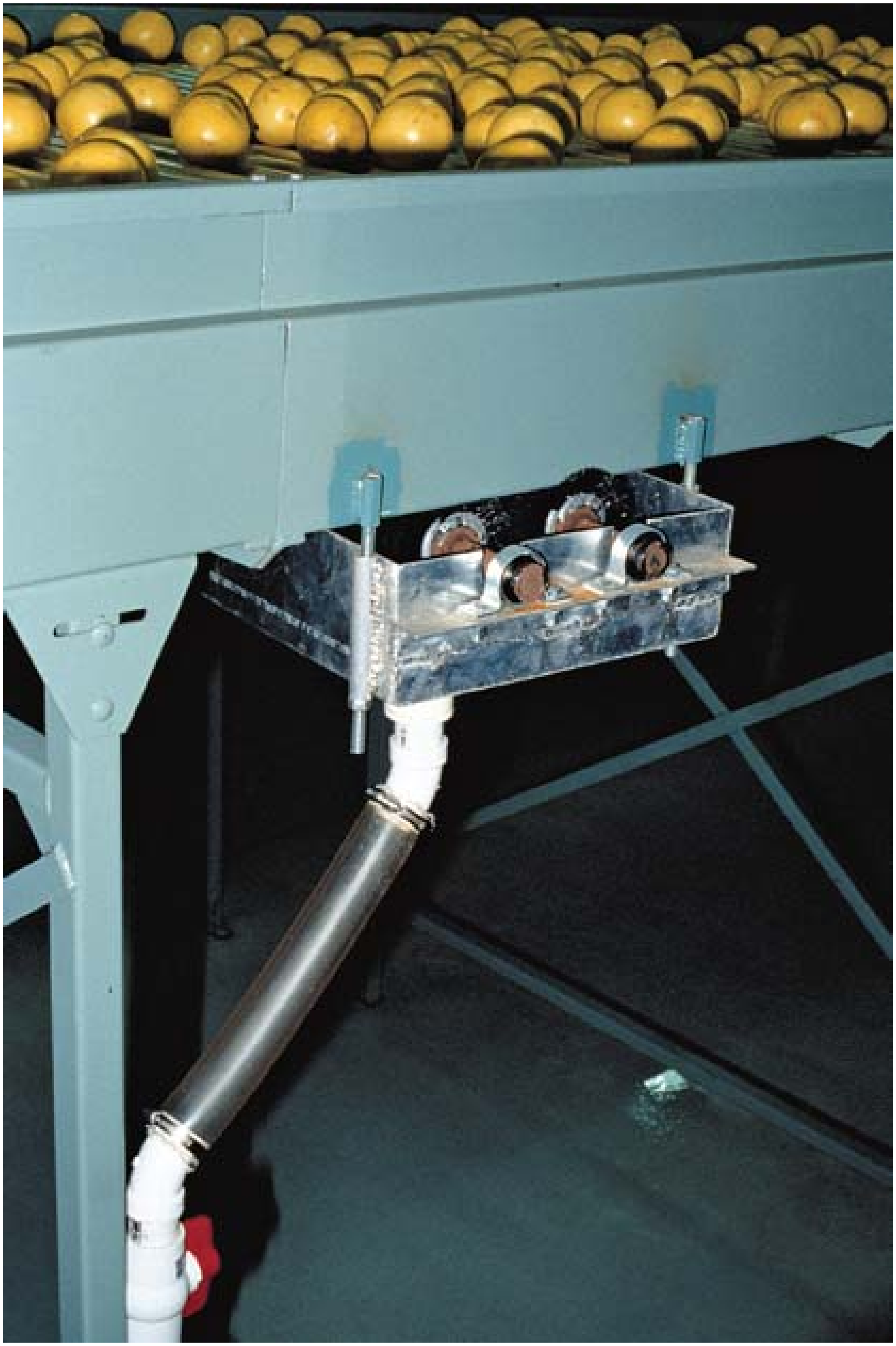


Figure 11.17. A cleaning unit with which a disinfestant is applied to fruit conveyor rollers for daily sanitation of the citrus packing line.

World Pesticide Use for Agriculture, 2000

- 44% herbicides
- 28% insecticides
- 19% fungicides

HERBICIDES

Herbicides kill plants. They are occasionally used to kill parasitic plants, but it is difficult to kill the parasite without harming the host. Weed reservoirs of plant viruses, fastidious bacteria, and certain other pathogens may be reduced using herbicides. Herbicides may be used to eradicate alternate hosts of rust fungi (Figure 11.18). Near the end of the growing season, herbicides are sometimes used to kill potato vines prematurely to prevent *Phytophthora infestans* (late blight) on the leaves from infecting the tubers. Herbicides are occasionally used to kill parasitic plants, but it is difficult to kill the parasite without harming the host.

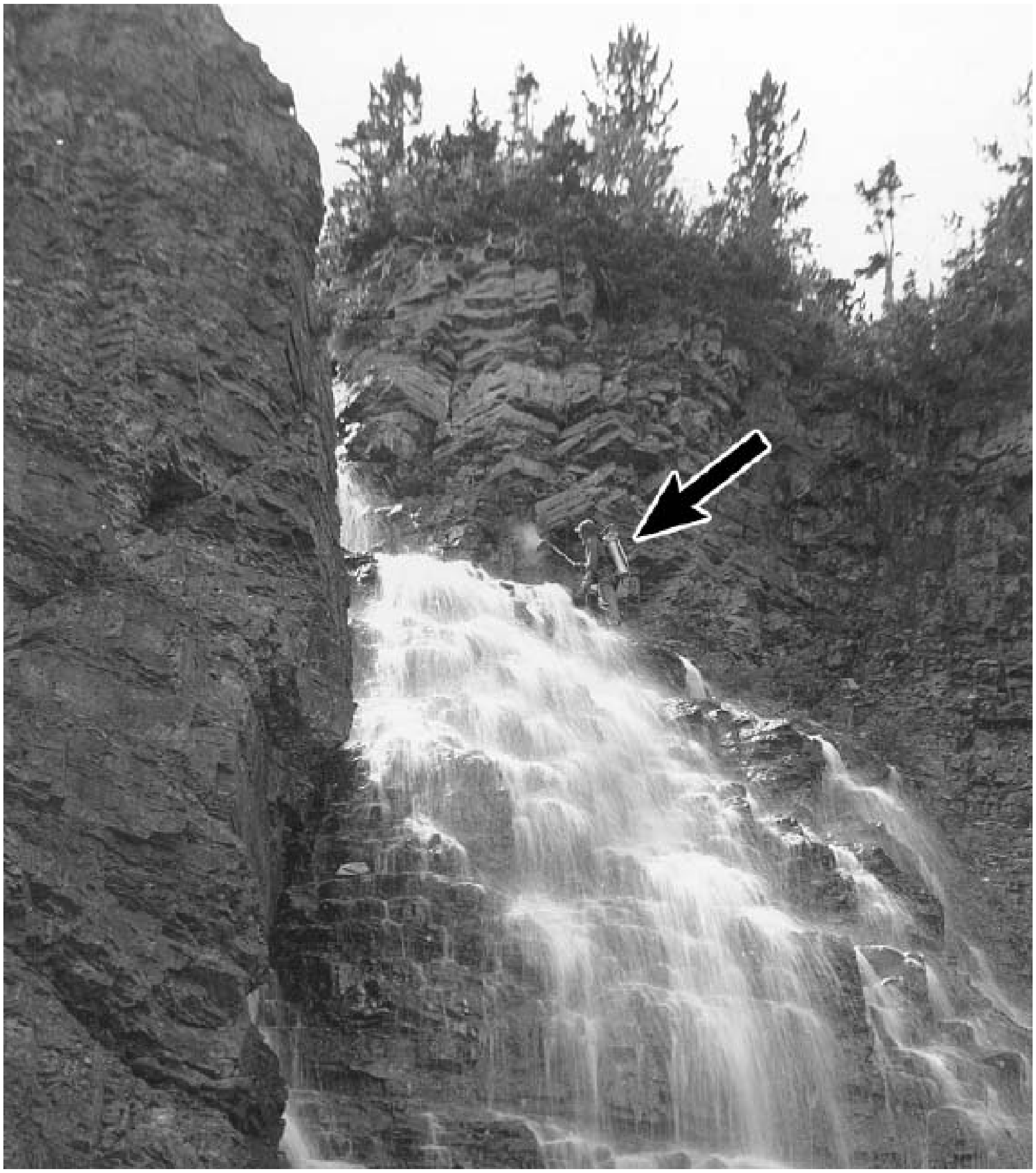


Fig. 11.18. Herbicide application to a *Ribes* bush, the alternate host of the white pine blister rust fungus, along a waterfall in Glacier National Park. The applicator is shown by the arrow.

INSECTICIDES

As discussed in previous chapters, the routine use of insecticides is not recommended to kill vectors of fastidious bacteria and viruses. In some cases, traps are used to monitor vectors to determine whether insecticide use is warranted. For example, in the United Kingdom, suction traps 12 m (36 ft) above the ground are used to monitor migrating aphid populations that may be carrying *Barley yellow dwarf virus*. The insects are tested by immunoassay to determine whether they are carrying the virus, and an insecticide is applied, if needed, to limit infections. Yellow sticky traps are commonly used in greenhouses to trap aphids and thrips, both of which can be vectors of harmful viruses (Figure 11.19).

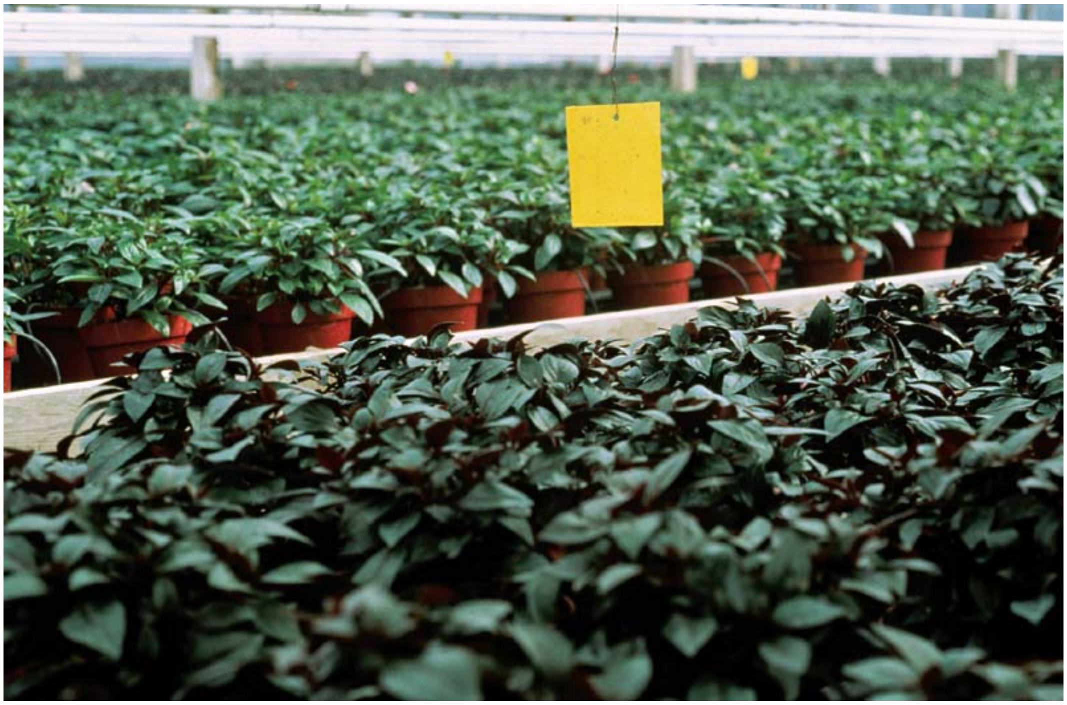


Figure 11.19. Yellow sticky cards may be used to monitor greenhouse crops for virus vectors such as thrips and aphids.

NEMATICIDES

These chemicals are used to reduce certain plant-pathogenic nematodes on high value crops. Most nematicides (e.g., aldicarb) target the nervous system, so they also kill soil insects, worms, and other beneficial organisms. Because of their toxicity, they must be used with particular care during application; their use is strictly regulated and may be banned in the near future.

Nematicide

- aldicarb

Soil fumigants

- chloropicrin
- dazomet
- 1,3-dichloropropene
- metam-sodium
- metam-potassium
- methyl bromide

SEED TREATMENTS

Some seeds are treated with surface fungicides to protect them from damping-off pathogens for a short time after planting. Chemical treatments with systemic fungicides are used to eradicate pathogens from seeds (Figure 11.20). Systemic fungicides are most commonly used for seeds of cereals that might be infected by various smut fungi (Figure 11.5). Copper and organic mercury compounds were used to treat seeds in the past, but they required soaking the seed in water or slurries and could be toxic to the seed embryos.

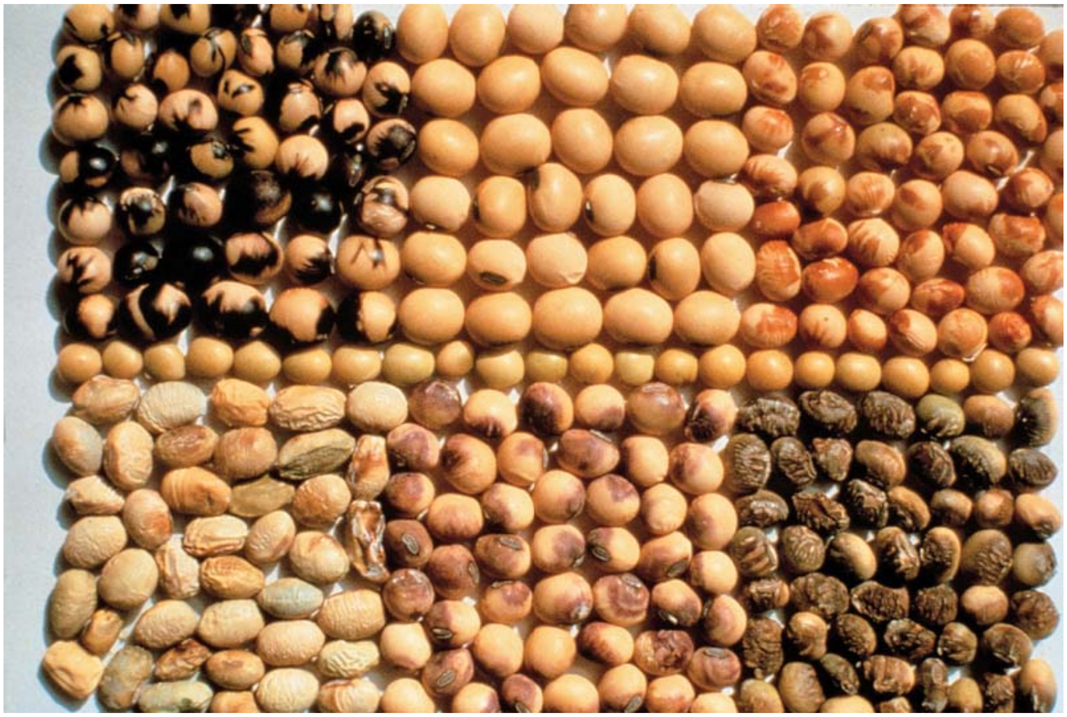


Figure 11.20. Soybean seeds from plants infected by a virus (upper left and right) or by several different fungi (lower rows). Seeds in the upper center area appear healthy.

SOIL FUMIGANTS

Fumigants were developed to temporarily reduce the populations of a variety of plant pests and pathogens. These chemicals harm plants, so the chemical must have time to dissipate after application and before seeding or planting. Some soil fumigants are so volatile that fields must be sealed with plastic tarps to retain the chemical in the soil long enough for it to be effective (see Figure 4.20). A number of soil fumigants are classified as “biocides,” which means that they reduce populations of fungi, nematodes, and weeds as well as many beneficial organisms. Some examples of soil fumigants are chloropicrin, dazomet, 1,3-dichloropropene, and methyl bromide. High cost limits the use of soil fumigation to high value crops or to perennial crops (e.g., fruit trees, golf putting greens, nursery crops, or strawberries) before plant reestablishment in soil with high pathogen populations. There are increasing concerns about applicator hazards and environmental problems, including destruction of the protective ozone layer (by methyl bromide) and groundwater contamination with toxic compounds. Some soil fumigants have been banned. The use of the remaining available compounds is strictly regulated.

Chemicals are used in a variety of ways to eradicate pathogens. Disinfestants are used for tools, pots, tires, and other inanimate objects that may carry pathogens. Herbicides are used to eradicate weed reservoirs of pathogens. Insecticides may be used to eradicate pathogen vectors. Nematicides eradicate plant-pathogenic nematodes but may affect other animals with a nervous system. Soil fumigation helps to eradicate most pathogens, weeds, and insect pests, but it is expensive and is restricted because of the toxicity of the chemicals used.

Biological Control

Modes of Action of Biological Control

For eradication of the pathogen

- parasitism (hyperparasites)
- predation
- hypovirulence

For protection of the plant

- antibiosis
- competition
- cross-protection

As discussed in Chapter 9, pathogens interact with numerous other microorganisms in their environment. **Biological control** is the deliberate exploitation of these microorganisms to reduce inoculum or protect plants. The concept of biological control grew out of careful observations of certain disease phenomena followed by investigations into their causes. For many years, **crop rotation** was thought to reduce host-specific pathogens by starving them in the absence of their host plants. We now know crop rotation could be considered biological control because competition with and antagonism by soil microorganisms reduce pathogen populations when rotation is practiced.

Suppressive soils, in which various diseases were naturally at lower levels than expected, have been observed. Further investigations demonstrated that the suppression is commonly due to biological factors in the soil, but, in most cases, it has not been possible to identify the exact causes and recreate these suppressive soils in other places.

► **DID YOU KNOW?**

The first attempts at direct applications for biological control of plant pathogens were made by C. Hartley in 1921. He inoculated pots of autoclaved soil with 13 antagonistic fungi to try to reduce damping-off of pine seedlings.

In some cases, disease declines when a crop is grown in the same soil for several years. With repeated planting of the same crop, disease first increases and yields decline for several years as expected, but then, surprisingly, some diseases begin to decline. **Take-all decline** in wheat is the best known example of this, but it also has been observed for other diseases such as necrotic ring spot, summer patch, and take-all patch of turfgrass. These diseases are more common and more severe in newly established turfgrasses and tend to decline as the turfgrasses mature. Research into the nature of disease declines has improved our understanding of the role of rhizosphere populations of microorganisms and how they function in biological control.

The previous examples demonstrate that general populations of soil microbes can help reduce diseases. **Green manures** are living crops that are plowed under to improve the soil but that also increase populations of antagonistic organisms, a form of general biological control. **Composts**, sometimes added as organic soil amendments, may reduce disease by introducing microbial populations (Figure 11.21). The successful use of compost depends on the organic base that is used and the types and numbers of microorganisms. Microbial colonization of compost occurs naturally after the thermophilic phase, and sometimes the colonizing populations do not suppress disease. Some commercial enterprises deliberately introduce specific organisms into composts to enhance their ability to suppress disease.

General Methods of Biological Control

Green manures: living plants plowed into the soil to stimulate the growth of populations of biological control organisms

Compost and other organic amendments: introduction of microbial populations and organic matter that helps to support microbial populations

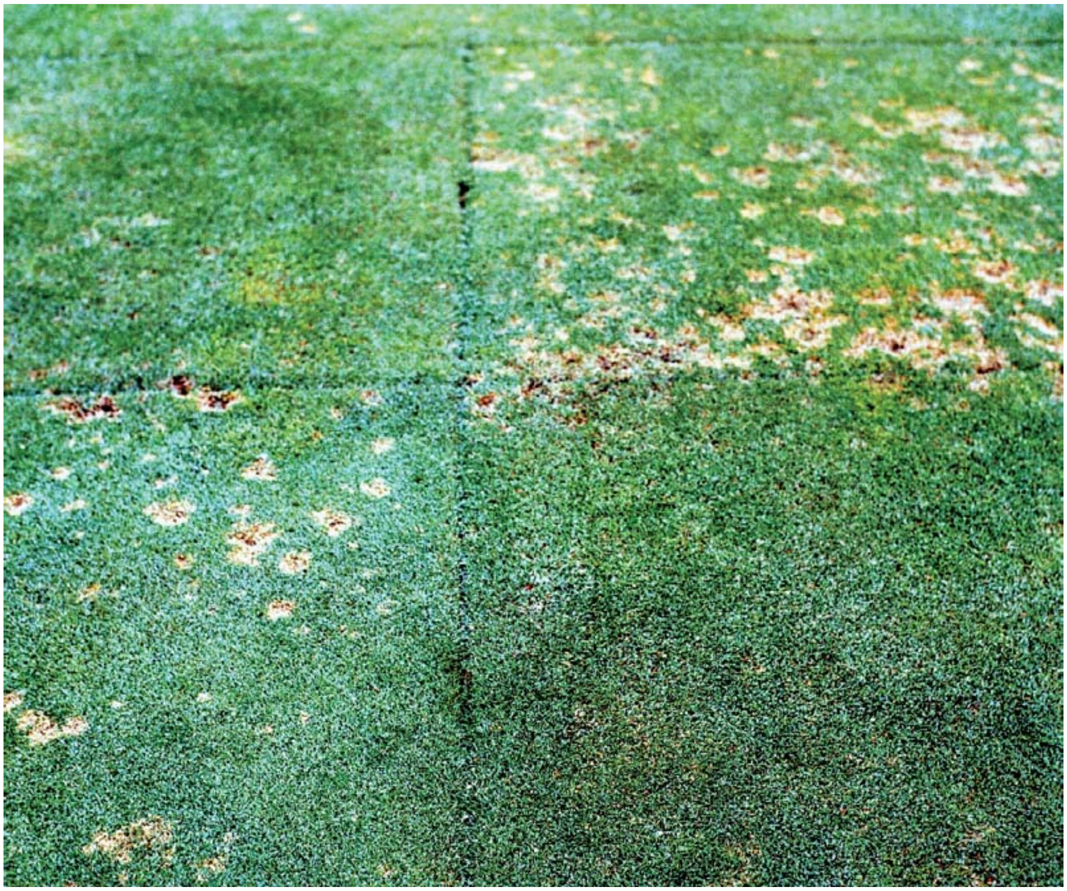


Figure 11.21. Reduction of dollar spot on turfgrass in plots to which compost has been applied (upper left and lower right).

All of these general enhancements of natural microbial populations that reduce plant diseases have led to the hope that specific biological control agents could be isolated and applied in ways similar to fungicides and other chemicals. As with chemicals, biological controls may be used to protect plants from infection or to eradicate primary inoculum. **Antagonist** is a general term for an organism that interferes with plant pathogens. Antagonists may act in various ways but have several specific modes of action. Two of these, **parasitism** and **predation**, mostly reduce inoculum and are considered forms of eradication. **Hypovirulence** describes a phenomenon in which the pathogen's ability to cause disease is weakened.

PARASITISM

Parasites of parasites are called **hyperparasites** (Figure 11.22A). They have been isolated from nematodes, nematode eggs, fungal survival structures, and mycelia. *Trichoderma* spp. are common soil fungi and among the most studied hyperparasitic fungi. Many other fungal and bacterial hyperparasites have been isolated from plant pathogens. It is sometimes difficult to produce and effectively distribute inoculum of hyperparasites, which has limited their use in commercial products.

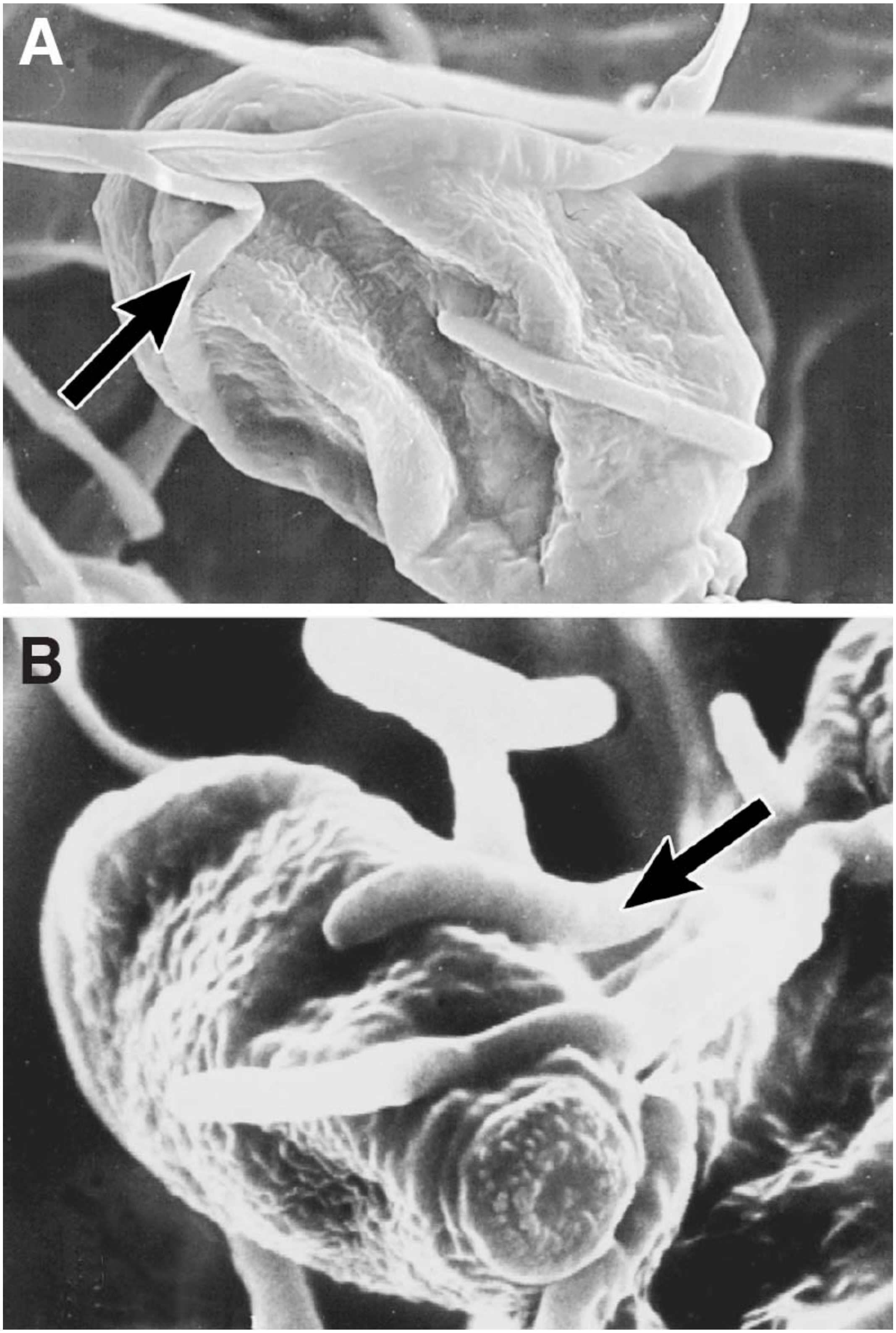


Figure 11.22. Fungi that offer biological control of powdery mildews using two different modes of action to attack the mycelium and conidia of the pathogen. A, *Verticillium lecanii* is a hyperparasite. Note that the

hyphae (arrow) penetrate and parasitize the conidium. B, *Pseudozyma flocculosa* (arrow) is an antagonist that causes collapse of fungal structures but never penetrates the powdery mildew fungus.

As with all parasites, it is important to know the host range of the organism. In some cases, a very narrow host range is a benefit because the hyperparasite is likely to infect only the pathogen and not beneficial organisms. Hyperparasites do not necessarily have to completely kill a pathogen to be effective. A fungal hyperparasite of a powdery mildew fungus was found to inhibit spore production and thus reduce the ability of the pathogen to spread (Figure 11.22A).

PREDATION

Various predaceous nematodes and other soil microfauna feed on plant-pathogenic nematodes and their eggs. Many nematode-trapping fungi have been isolated from soils (see Figure 4.22). Commercial products utilizing predation for control of pathogens are still in the research and development stages.

HYPOVIRULENCE

This phenomenon is best known in chestnut blight (see Robert Frost's prediction in Chapter 10) (Figure 11.23). A **mycovirus**, a virus that infects the pathogenic fungus, reduces the virulence, or disease-causing ability, of the pathogen. The hyphae of genetically compatible strains of the fungal pathogen may fuse (anastomose), and the mycovirus can then spread from the hypovirulent strain into the mycelium of the virulent strain, reducing its ability to cause disease. These hypovirulent strains are applied to active chestnut blight cankers to restrict their growth. This has been accomplished successfully in chestnut orchards in Europe, where there are relatively few genetic strains of the pathogen. The problem is more challenging in the forests of the Appalachian Mountains, the natural habitat of the American chestnut. The extensive geographical range and the numerous genetic strains of the pathogen have limited the successful use of hypovirulence so far. Mycoviruses have been found in other fungi, but practical uses have not yet been developed.

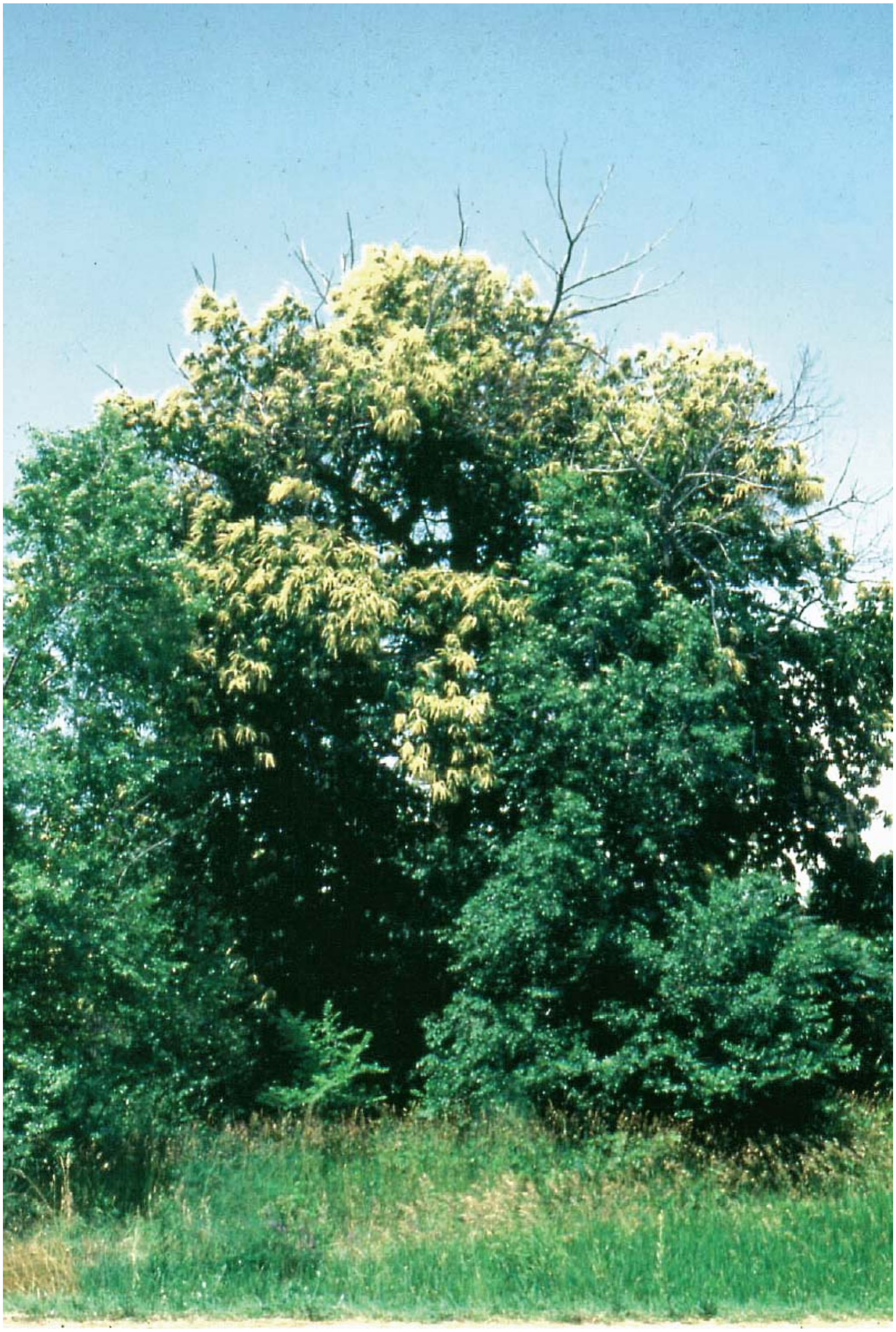


Figure 11.23. A chestnut tree in Michigan recovering from chestnut blight because of naturally occurring hypovirulent strains of the pathogen.

► **DID YOU KNOW?**

The American chestnut might return a century after the terrible chestnut blight began with the introduction of the ascomycete *Cryphonectria parasitica*. By 1950, 3.5 billion trees, nearly 90% of the U.S. population of chestnuts, had died. The American chestnut was sometimes called the “redwood of the East” and was important as a source of decay-resistant lumber, tannins for the leather-tanning industry, and, of course, its delicious nuts. The nuts not only were important to people who collected them for food and income but also were an important food for bears, wild turkeys, and other animals of the forest. Several groups of scientists are working to bring back the chestnut in its natural range in the Appalachian Mountains of the eastern United States. Traditional plant breeding and genetic engineering are being used to create a variety of blight-resistant chestnut trees. Other scientists are trying to use the phenomenon of hypovirulence, which results when a pathogenic fungus is infected by a virus and weakened. As with all plant diseases, a combination of approaches is most likely to succeed.

Careful observations of plant disease epidemics provided evidence that biological control of plant diseases might be possible. The effectiveness of crop rotation, suppressive soils, and disease declines were studied and found to have a biological basis. Green manure and compost amendments are used to generally increase populations of antagonists. Biological controls can be eradicative or protective. Hyperparasites and predators have been isolated and used to

reduce pathogens and their inoculum. Hypovirulence has been used to stop the expansion of chestnut blight cankers and may be useful against some other pathogens.

How do we protect plants?

The previous sections described methods to avoid, exclude, or eradicate pathogens. These are useful for both monocyclic and polycyclic pathogens. They are the primary methods used to manage monocyclic pathogens, for which primary inoculum is most important. Although it is helpful to reduce the amount of **primary (initial) inoculum**, another option is to reduce its efficacy, i.e., to **protect** the plant from infection by primary inoculum even if it is there. For polycyclic pathogens, it also is necessary to protect plants from **secondary inoculum** for as long as the plants remain susceptible to infection. Luckily, there is a wide range of options to protect plants against infection. They can be grouped as cultural practices, chemicals, biological controls, and genetic resistance. Not all of these can be used on every kind of plant or for every kind of pathogen, but usually several them will be generally effective in reducing disease.

Methods of PROTECTION

Cultural practices

- barriers and mulches
- plant nutrition
- modification of soil pH
- temperature management
    - cool plants
    - cold storage of plant products

- water management
    - improve soil drainage
    - minimize leaf wetness

Chemical protection

- for bacteria
    - antibiotics
    - copper-based chemicals
- for fungi
    - protectant/contact fungicides
    - systemic/penetrant fungicides

Biological control

- antibiosis
- competition
- cross-protection

Genetic resistance

- specific (R gene) resistance
- general resistance

Cultural Practices

> “Cultural practices are often forgotten or barely mentioned in the modern plant disease literature, even though many farmers have successfully managed plant diseases for thousands of years, primarily with cultural practices.”
>
> —H. D. Thurston, 1992, *Sustainable Practices for Plant Disease Management in Traditional Farming Systems*

BARRIERS AND MULCHES

It is sometimes possible to create barriers that help to protect plants. In greenhouses, it is common to use very fine mesh screens that prevent access of insect vectors of fastidious bacteria or viruses. Outdoors, reflective or colored plastic mulches are sometimes used to repel aphids and other vectors. Unfortunately, as plants grow larger, they tend to cover the mulch and reduce its effectiveness, but mulches can delay infection substantially and thus increase yields. Organic and plastic mulches also can help to protect the "splash zone" parts of plants such as tomatoes and cucurbit fruits from soilborne pathogens such as oomycetes (Figures 11.24 and 11.25). Shredded bark and other organic mulches serve as physical barriers to foliar pathogens, including nematodes attempting to move upward from the soil and plant debris on the soil surface.

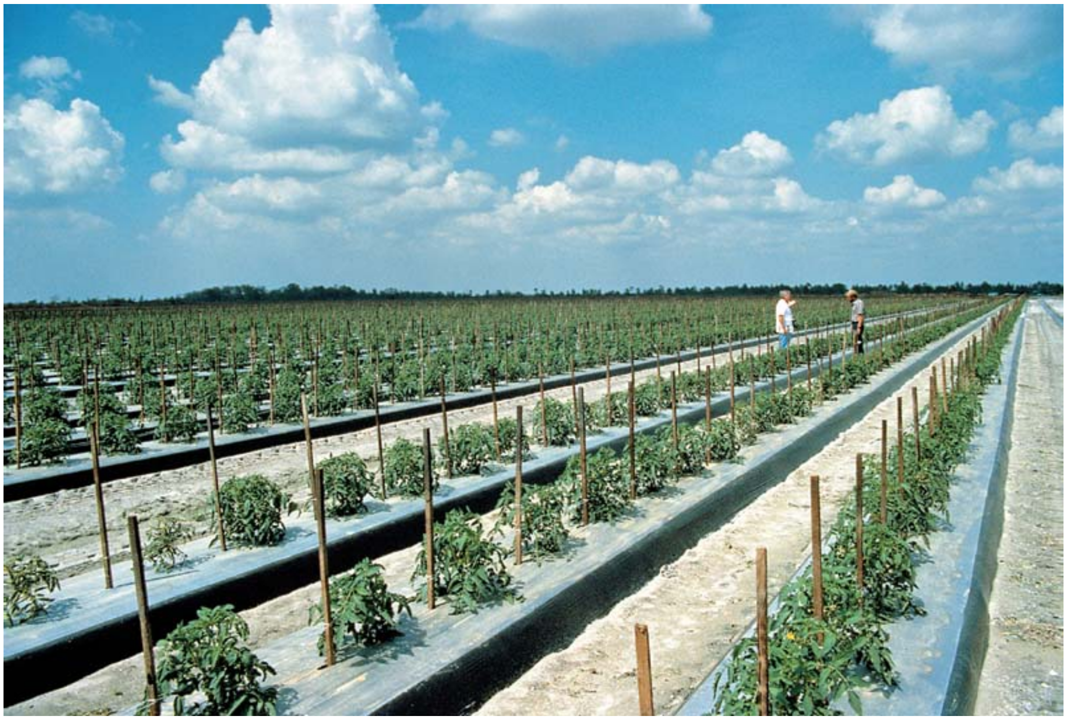


Figure 11.24. Tomatoes staked and growing on plastic mulch to aid drying of plant surfaces and to protect plants from soilborne pathogens that might splash up from the soil.

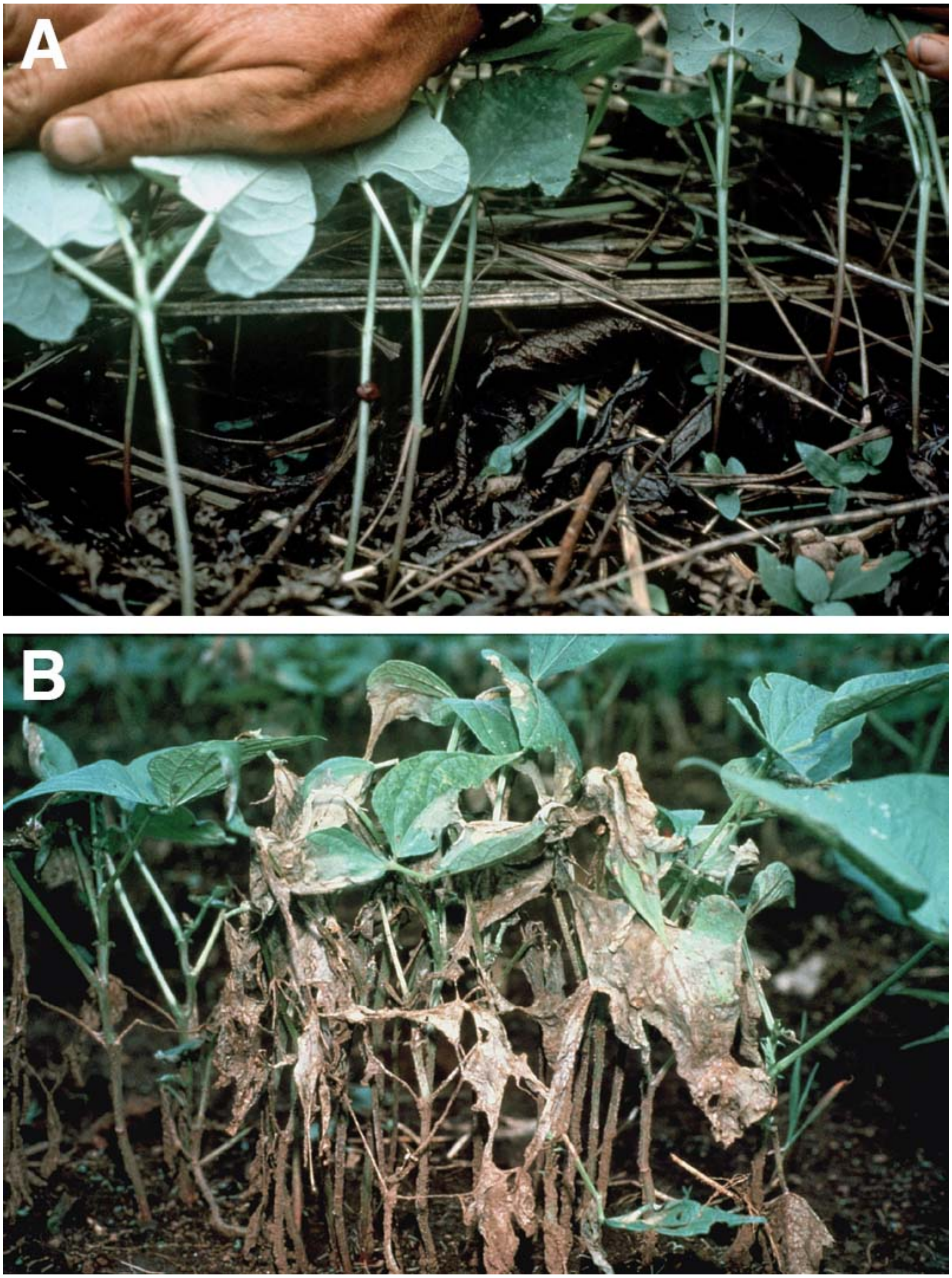


Figure 11.25. Protection by a barrier. A, Use of organic mulch (a method called *frijol tapado*, "covered beans") as a barrier to the soilborne fungus that causes web blight of beans (*Thanatephorus cucumeris*). The mulch also reduces weed growth and conserves soil moisture. B, Web blight of beans where organic mulch was not used.

PLANT NUTRITION

As discussed previously, nutrient deficiencies can stress plants and make them more susceptible to diseases. The macronutrient nitrogen seems to have the most important impact on plant diseases by causing lush tissue when used in excess and senescent tissue when deficient. A combination of small applications of quick-release nitrogen fertilizers along with a base of slow-release nitrogen helps to prevent the extremes that can enhance diseases. Frequent small applications of fertilizer (“spoon-feeding”) often are recommended for greenhouses and golf courses. In some systems, fertilizers are applied in the irrigation water, a process called “fertigation.”

SOIL pH

Recommendations for soil pH level are generally based on plant requirements and the effects on the availability of nutrients. Certain plants, such as ericaceous species (blueberry, rhododendron), grow best in acid soils. Soil pH is sometimes modified to reduce infection by certain soilborne pathogens. Some classic examples are raising pH to about 7.2 to reduce clubroot of brassicas (crucifers) and maintaining pH at 4.0–5.2 to reduce common scab of potatoes. Necrotic ring spot, summer patch, and take-all patch of some turfgrasses are reduced by lowering soil pH with the use of sulfur or certain forms of nitrogen fertilizer such as ammonium sulfate. Severity of Verticillium wilt is often higher in soils with pH levels above 7. Most soilborne pathogens are not affected by soil pH within the range appropriate for plant growth.

TEMPERATURE

Temperature is often a determining factor in disease severity. For example, *Sclerotium rolfsii* causes “southern blight” on a wide variety of herbaceous and woody plants in hot, humid climates. Snow mold fungi on turfgrass and cereal crops are active primarily in cool, humid conditions, such as under snow cover. Pythium blight is a foliar disease of turfgrass in hot, humid weather, but Pythium root rot often occurs in cooler conditions.

Temperature is controlled in greenhouses with **shade** and ventilation by **fans. High tunnel vegetable production** is a simplified growing system that uses a single layer of polyethylene to form a tunnel-shaped cover over a crop and its irrigation system. Unlike a conventional greenhouse, it uses no electrical components for light or air movement. The cover protects the crop from temperature fluctuations and frost and thereby extends the growing season. It also reduces the length of time that leaves are wet, so foliar diseases are minimized (see below).

For some plants, midday heat can be dissipated by brief applications of irrigation water, known as **syringing** when used on golf course putting greens. **Cold** is used to reduce pathogen activity in stored harvested crops, seed, and propagative parts.

WATER MANAGEMENT

Poorly drained soils increase root diseases by reducing the oxygen supply to roots and enhancing the activity of many pathogens, especially oomycetes (Figure 11.26). Improved drainage reduces problems associated with some pathogens. Although compacted, poorly drained soil is an important factor in many soilborne diseases, it also increases the humidity around the aboveground parts of plants and lengthens drying time.

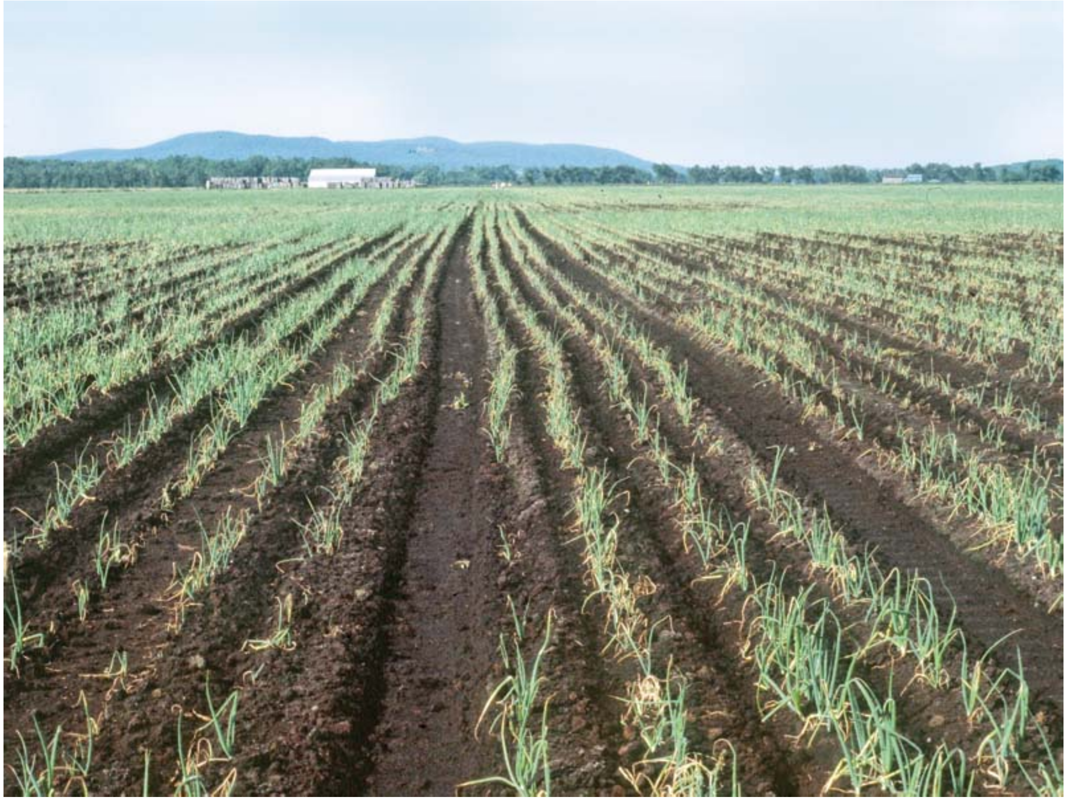


Figure 11.26. Damage to onions caused by *Pythium* spp. in a field with poor drainage.

Leaf wetness is a key factor for foliar infection by bacteria, fungi, and foliar nematodes. Leaf wetness can be reduced by several practices.

Methods of Reducing Leaf Wetness

- fans
- improve soil drainage
- plant spacing and form
- removal of dew
- row orientation
- shade reduction
- soil surface irrigation
- timing of irrigation
- weed control

1. Fans. Fans move air, increase evaporation, and thereby reduce leaf wetness. They are often used in greenhouses and on some golf courses.

2. Plant spacing and form. Use of the recommended spacing of herbaceous plants helps to reduce competition among plants and helps plants dry quickly as they mature and the leaf canopy closes. On woody plants, thinning of canopies by pruning branches enhances air movement and drying.

3. Removal of dew. It is best to mow lawns after the turfgrass leaves are dry to avoid spreading pathogens. However, early morning mowing of golf course putting greens and fairways is usually a commercial necessity (Figure 11.27). On turfgrass with very short leaf blades, mowing can reduce foliar diseases by removing dew and nutrient-rich guttation fluid because the leaf blades dry quickly. On days when the greens and fairways are not mowed, hoses or poles may be dragged across the surface of the turfgrass to remove dew and reduce the length of time that the leaves remain wet.

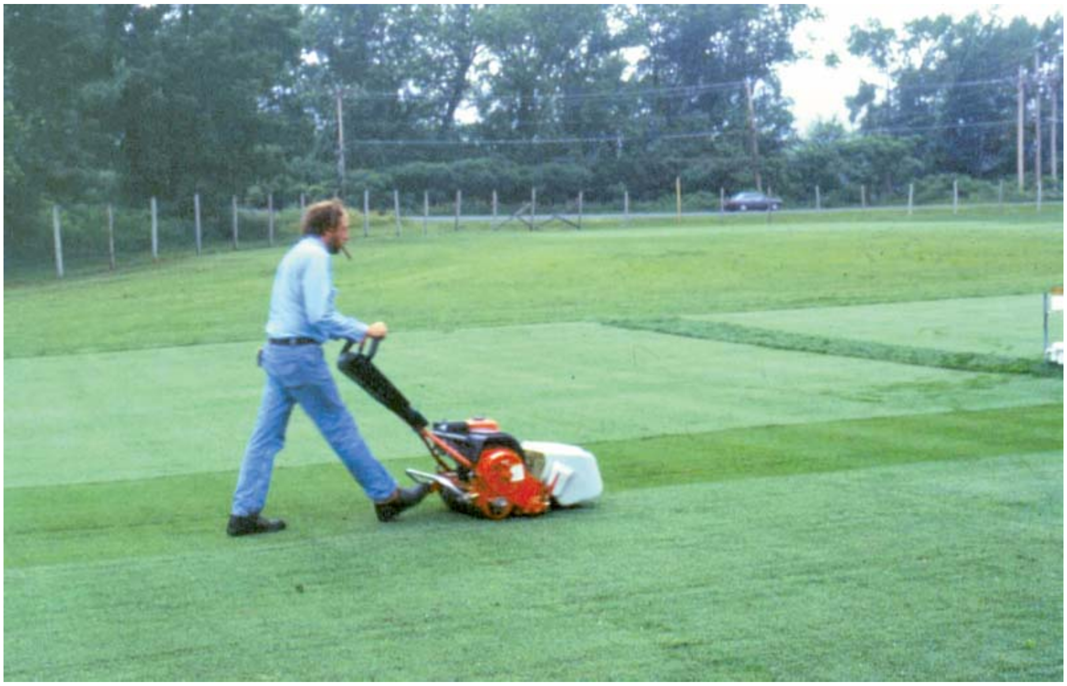


Figure 11.27. Removal of dew on turfgrass by early morning mowing.

4. Row orientation and air movement. At the time of planting, rows should be oriented to take advantage of prevailing air movement patterns, so leaves will dry quickly. Hedgerows along field crops and plantings on golf courses and in landscapes can impede air movement and allow retention of moisture on plants (Figure 11.28).

Figure 11.28. Trees bordering this potato field may impede air movement and slow the drying of plants.

5. Shade. In outdoor plantings, areas may be in shade for parts of the morning or afternoon. Morning shade, in particular, increases the time that leaves remain wet from dew that forms overnight. Outdoor plants that are shaded for much of the day often have more foliar diseases, such as powdery mildew, because of the increased humidity in their environment. Shading also reduces ultraviolet light damage to pathogens that are exposed on plant surfaces, such as powdery mildew fungi.

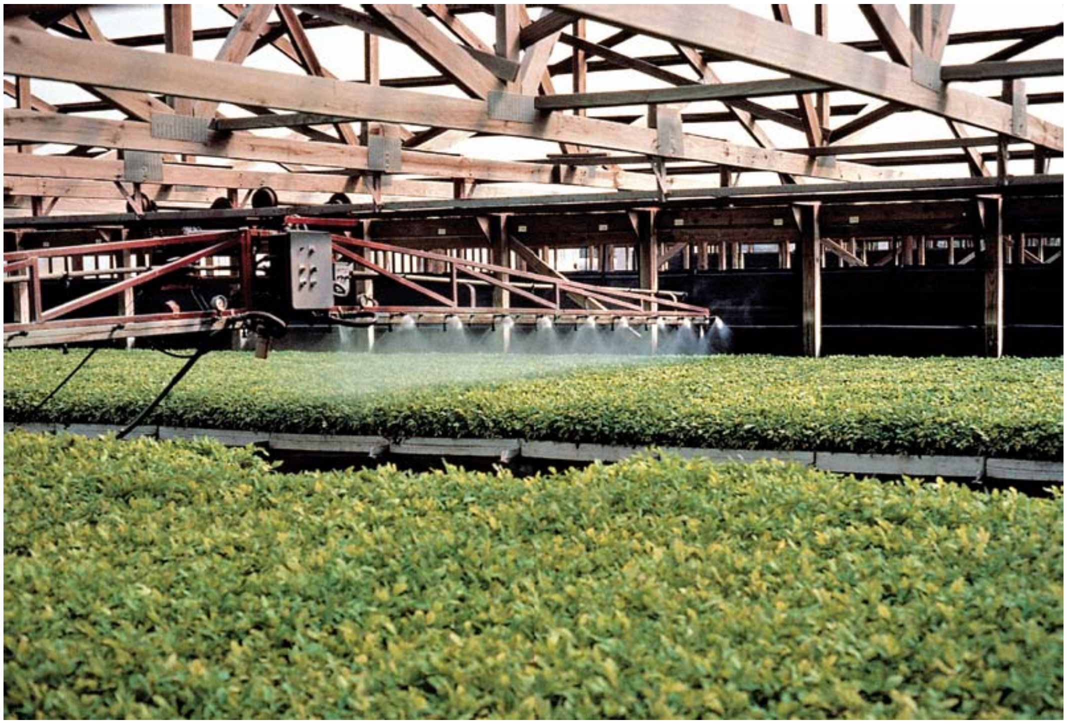


Figure 11.29. Overhead irrigation and fertilization (fertigation) of greenhouse-produced tomato transplants. This can potentially contribute to pathogen dispersal and leaf wetness.

6. Soil surface irrigation. In greenhouses and some outdoor plantings, irrigating only the soil surface greatly reduces foliar diseases by minimizing periods of leaf wetness (Figure 11.29).

7. Timing of irrigation. Water should be applied in the early morning when the leaves are usually already wet with dew. Water also can be applied during the day when leaves have time to dry quickly, before infections can occur. Water conservation programs discourage midday watering because so much water is lost to evaporation. Watering in the late afternoon and early evening, or just as the leaves are drying in the morning, extends the period of leaf wetness and increases the potential for disease.

8. Weed control. Weeds can be a barrier to rapid drying of desirable plants.

To protect plants, cultural practices should favor plant growth and reduce opportunities for infection by pathogens. Physical barriers to pathogens and vectors such as greenhouse screens and mulches are sometimes effective. Temperature, soil pH, and plant nutrition affect both plants and pathogens. Because so many bacterial and fungal foliar pathogens require hours of leaf wetness for infection, many cultural practices focus on minimizing this time.

Chemicals

Chemicals that are used to reduce primary inoculum were discussed previously and include soil fumigants, nematicides, insecticides for vector populations, herbicides for pathogen reservoirs in weeds or management of parasitic plants, seed treatments, and various disinfestants. The most common chemicals used to protect plants are **antibiotics** and copper compounds for bacterial pathogens and fungicides for fungal pathogens. “Plant activators” are the newest crop protectants. They induce **systemic acquired resistance (SAR)** to a variety of pathogens (see Chapter 9, Section B).

CHEMICALS FOR TREATMENT OF BACTERIAL DISEASES

Because of their expense and the risk of bacteria developing resistance, antibiotics are not used very often for bacterial plant diseases. In fact, only 0.1% of the antibiotics used in the United States are used for plant diseases. The antibiotics streptomycin, tetracycline, and oxytetracycline are sometimes applied to apples, pears, and some ornamentals to manage fire blight and other bacterial diseases. Tetracycline antibiotics may be injected into trees infected with phytoplasmas or *Xylella fastidiosa* for remission of symptoms, but they do not cure the trees.

Copper-based fungicides are used to manage the bacteria that cause citrus canker and foliar bacterial diseases such as bacterial spot of peppers and tomatoes. Copper formulations remain on the surface of the plant, so they are strictly protective, i.e., they offer no after-infection activity. They must be reapplied frequently to new tissue or when rain dilutes the application. Repeated applications can lead to a buildup of copper in the soil, which can cause toxicity in plants.

Copper-Containing Sprays

- strictly protective (no after-infection activity)
- used for fungal and bacterial diseases
- must be reapplied every 5–10 days, depending on rainfall
- copper may build up in soil with repeated use

Bordeaux mixture (copper sulfate and lime) was the first effective foliar fungicide that could be sprayed on plants to prevent infection by fungi. It was named by Alexis Millardet, a professor at the University of Bordeaux, in France. A French farmer created the original mixture to leave a chemical residue on grape vines along the roadside to discourage pilfering of his grapes. Millardet noticed that the sprayed vines were not dying of the new downy mildew disease that was threatening the French wine industry.

Plant Disease Profiles: Grape downy mildew

► DID YOU KNOW?

During World War I in Germany, copper was confiscated from citizens by the German army for military uses such as shell casings. This led to a shortage of the copper sulfate necessary for Bordeaux mixture, the only effective foliar fungicide at the time. Subsequent late blight epidemics of potatoes caused by *Phytophthora infestans* led to starvation of many German people and weakened the morale of their soldiers at the fronts.

FUNGICIDES

The earliest fungicides were inorganic compounds based on the toxic elements cadmium, copper, mercury, and sulfur. Cadmium and mercury fungicides are no longer available because of their toxicity to humans and the hazards they posed to the environment. Copper- and sulfur-based fungicides are effective against a wide range of fungal diseases, but they are strictly protective, must be reapplied often, and may accumulate in the soil. Organic fungicides (carbon-based compounds) tend to be more specific in their mode of action, so accurate diagnosis of the problem to be treated is more important.

Every year, plant pathologists test fungicides, and many publish their results in the online journal, *Plant Disease Management Reports*. Extension specialists and crop consultants develop these results into recommendations. Fungicide recommendations change often, and the availability of fungicides varies from country to country and from state to state. Certain fungicides may be used on turf and ornamental plants only, and others are used on food crops only. Overuse of many of the organic fungicides available today can result in resistant fungal populations, so it is important to use fungicides as part of an overall resistance management plan. Thus, there is no simple answer to the common question: “What is the best fungicide for this disease?”

DISEASE CLASSIC

Black Sigatoka of Banana and Plantain

Hosts banana and plantain

Pathogen *Mycosphaerella fijiensis* (an ascomycete)

Symptoms and Signs The disease begins as a leaf spot, but can cause large areas of blackened and water-soaked leaf tissue filled with pseudothecia of the pathogen that contain asci with two-celled ascospores. Sporodochia with long, septate conidia form on the lower leaf surface.

Disease Cycle Ascospores are the primary inoculum for long-distance dispersal and most secondary cycles. Spores infect through stomata in warm, wet weather. Because of the climate where bananas are grown, this can be almost a continuous polycyclic disease.

Management Large plantations rely mostly on fungicides. For small farmers, resistant cultivars are very important; however, breeding bananas is difficult because many (sterile triploids) do not produce seeds. Some cultural practices that help reduce leaf wetness, such as spacing of plants, can reduce disease.

Significance This disease can reduce yields by 50% and can require 38–50 fungicide applications each year. Black Sigatoka threatens an important food crop for many traditional farmers in tropical countries throughout the world.

Plant Disease Profiles: Black Sigatoka of bananas and plantains

► DID YOU KNOW?

Bananas are fourth among agricultural commodities, with world trade of $2.5 billion annually. Yet, 90% of all bananas grown are consumed by poor subsistence farmers in tropical Africa, America, and Asia (Figure 11.30). The fungal disease, black Sigatoka, is responsible for 15-20% of the costs of bananas in importing countries, because 38–50 fungicide sprays may be applied each year to manage this disease.

Figure 11.30. Market in Malawi where several banana cultivars are for sale by traditional farmers.

Fungicides are used to protect high value crops from polycyclic diseases. They also are used for monocyclic diseases in which primary inoculum remains available for long time periods or for which other means of control are not available (Figure 11.31). Most crops do not get routine applications of fungicides because we have cultivars with good genetic resistance or cultural practices that effectively manage the disease. Fungicides are not used for most root diseases because it is often too late for treatment to be effective when symptoms are noticed. It also is difficult to deliver fungicides to roots effectively.

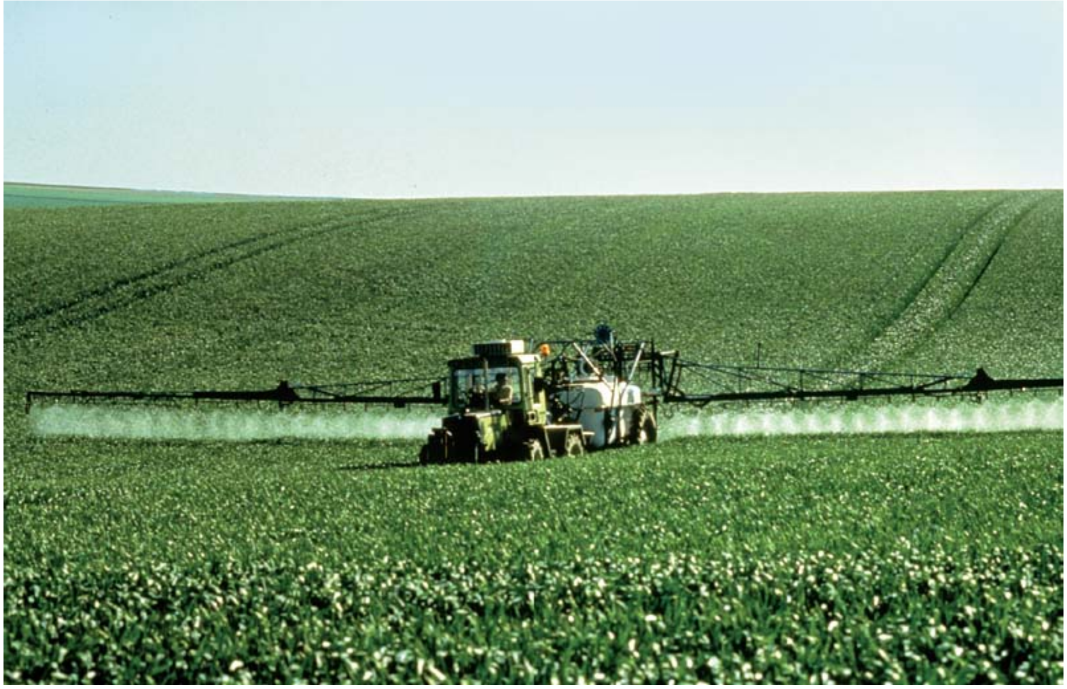


Figure 11.31. Application of fungicide to a potato field.

Fungicide Use in the United States

- mostly in the eastern half, where the climate is humid, and in humid parts of other states such as Oregon and Washington
- most useful for:
  - polycyclic pathogens
  - foliar pathogens
  - high value crops or crops susceptible to a disease that we cannot control in other ways (e.g., late blight of potato)
- 94% of fungicides are applied to fruits and vegetables, including potatoes.
- 50–65 million pounds of active ingredient are applied each year.

Organic fungicides are toxic to fungi, but most of them are not particularly toxic to humans and other nontarget organisms. A few fungicides are toxic to fish or other organisms. Specific toxicity warnings are found on the fungicide label.

The **relative acute toxicity** of a substance is usually determined by calculating the dose that is lethal for 50% of a test population, often rats or mice. This is called the median lethal dose (**LD_{50}**). It is stated as the amount of active ingredient (in milligrams) per kilogram of body weight. The LD_{50} is chosen as the halfway point in a toxicity test because certain individuals in any population are very sensitive to the substance, and others may be quite resistant. LD_{50} is a relative measure used to compare the toxicity of one compound to that of another. It is not a measure of exactly how much would be lethal to the applicator. LD_{50} also is a measure of acute exposure, i.e., a single dose all at once, but not of the chronic (low-dose and repeated) exposures that most applicators face.

Most fungicides have a high LD_{50}. This means that the lethal dose is quite large, and the product is not very toxic. This does not mean that there is no risk to the applicator, however, because chronic exposure or exposure to high concentrations of many chemicals can potentially cause other health problems, such as cancer. An LD_{50} usually represents an oral dose, but some chemicals can be absorbed very well through the skin and lungs, so appropriate protective clothing, gloves, and respirators should always be used.

Median Lethal Dose (LD_{50}), a Measure of Acute, Relative Toxicity

Substance	Amount (mg/kg of body weight)
aldicarb (nematicide/insecticide)	1

nicotine (insecticide)	50
caffeine	250
carbaryl (insecticide, e.g., Sevin)	307
aspirin	780
chlorothalonil (fungicide, e.g., Bravo, Daconil)	3,800
fenarimol (fungicide, e.g., Rubigan)	10,000

Nearly all of the fungicides used today are organic compounds that are eventually degraded by bacteria. Fungicides vary in their modes of action (i.e., how they work) and in what happens when they are applied to plant tissue. Factors that should be considered when choosing a fungicide are the following.

1. Application. Fungicides are most easily applied to aboveground plant parts. Most fungicides are formulated to be mixed in a water carrier and sprayed on aboveground parts of plants. A few fungicides are formulated as granules. It is difficult to apply fungicides to roots and other belowground parts. Only one kind of fungicide (phosphites such as fosetyl-Al), can actually move downward in the plant following a foliar application. Therefore, root diseases almost always require that a fungicide be drenched in with large volumes of water. In addition, by the time aboveground symptoms of a root disease are noticed, considerable damage has already occurred, and it is probably too late to stop the pathogen with a fungicide.

Limitations to the Use of Fungicides for Root Diseases

- Delivery to the root system is difficult.

- Nearly every fungicide must be watered in (only phosphites, e.g., fosetyl-Al, move downward after a foliar spray).
- It is usually difficult to know that a fungicide is needed until it is too late.

2. Pesticide label and food safety. It is important to obey all pesticide label instructions because they constitute a legal document determined at the time of registration with the U.S. Environmental Protection Agency (EPA). The label includes dose, frequency of application, which plants may be treated, and other information. To reduce worker exposure, intervals may be required after an application before reentry is allowed. Labels for fungicides and other pesticides applied to food crops include a **days to harvest** time to minimize residues in food products. For example, if the label indicates five days to harvest, the crop may not be harvested sooner than five days after the last fungicide application. The days to harvest are calculated from toxicological studies of safe exposure levels for a pesticide (plus a safety factor) as well as the time it takes for a pesticide to attenuate after application (plus a safety factor). The goal of the calculation is to minimize residues at the time of harvest. A **tolerance**, or maximum legal residue, is established for all food-crop uses of pesticides. Any crop that has residues in excess of the tolerance can be confiscated and destroyed. Major food processors do their own testing, and representatives from the EPA can test any harvested crops for residues.

► DID YOU KNOW?

Each year, the U.S. government tests domestic and imported fruits and vegetables for pesticides. In a typical year, over 14,000 samples were tested. Of these, more than 50% had no residues, and only 1.5%

exceeded the tolerance. The total dietary intake of pesticide residues is <1% of the acceptable daily intake (ADI).

In the *Annual Review of Phytopathology* in 2000, Dr. Nancy Ragsdale examined the impact of the Food Quality Protection Act (FQPA) on the future of plant disease management. She wrote: “Public perception and political pressure were sufficient for the U.S. Congress to unanimously pass FQPA although scientific data indicating that our food supply was unsafe because of pesticide residues were lacking.”

In 1996, the **Food Quality Protection Act (FQPA)** was passed by the U.S. Congress. One of its goals was to apply the science of food safety to the registration of pesticides. It considers all potential exposures to a particular pesticide, including uses in recreational areas and home pest control as well as food residue exposures. It also looks at pesticide exposure by chemical groups with the same mode of action and therefore the same health impacts on people. These are combined into what is called a **risk cup** that represents the total exposure of a given pesticide group that a person could be exposed to every day, for 70 years, without additional health risks. If the residue exposure exceeds what the risk cup allows, then exposure must be reduced.

Chemical companies must then eliminate uses based on potential exposure and the economic value of the uses in product sales. For example, a number of fungicide registrations for lawn care have already been eliminated because the potential pesticide exposure is high near homes and the economic value of these uses to the companies is relatively low.

3. Spills and care in application. Care should be taken to avoid spills or inappropriate disposal of concentrated amounts of fungicides and other pesticides in containers. Sprayers should be calibrated, spraying near surface and ground waters (e.g., wells and high-risk areas) should be avoided, and applications should not be made in high winds or before impending rainstorms.

In recent years, the contamination of groundwater by pesticides has become a concern (Figure 11.32). Groundwater is especially vulnerable to contamination (whether by septic tanks, buried gas tanks, or pesticide applications) in sandy soils with low organic matter, where groundwater is close to the surface, and where the climate is wet or irrigation is heavy. Pesticide characteristics also play a role in groundwater contamination. Chemicals are at a greater risk for movement into groundwater if they are soluble, do not adsorb well to organic matter, are applied to bare soil, or are injected or watered into soil.

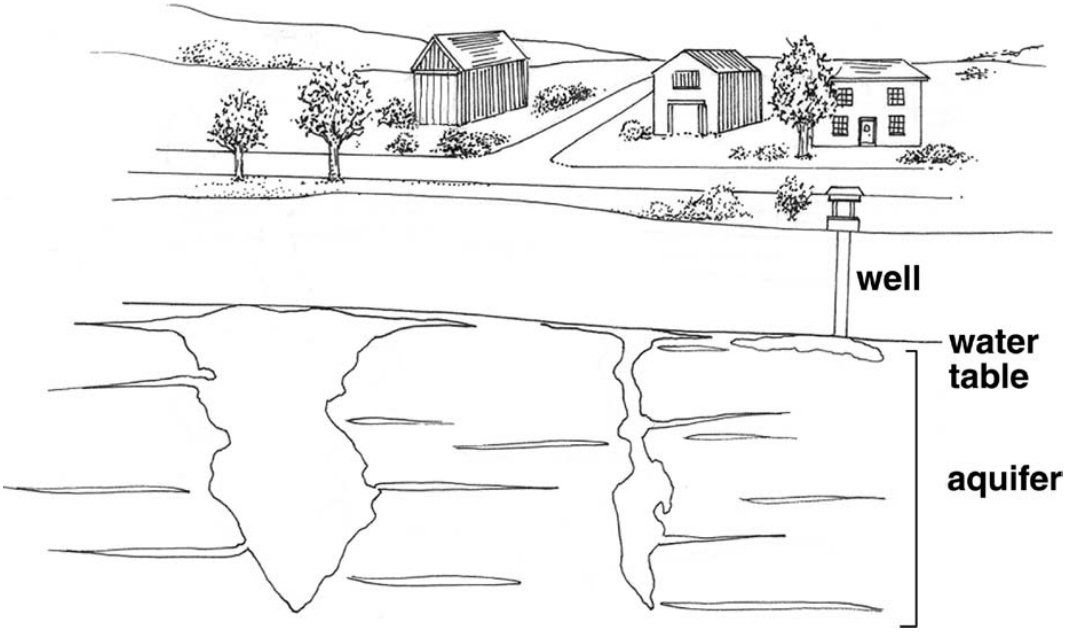


Figure 11.32. Diagrammatic view of groundwater sources in aquifers.

Groundwater contamination has occurred in Cape Cod (in Massachusetts), Florida, Long Island (in New York), and some sandy river valleys in the eastern half of the United States. The most common pesticide groundwater contaminants are preemergent herbicides, soil fumigants, and nematicides—all chemicals that are applied on or into bare soils. When fungicides are applied to growing plants, potential contaminants are more likely to be degraded by the increased microbial activity of the rhizosphere before they reach the groundwater. This is one reason that grass plantings, with their dense root system and organic thatch layer, help to prevent groundwater contamination. Grasses are sometimes planted between rows in perennial plantings to protect groundwater or at the edges of agricultural fields to help protect surface waters from run-off contamination.

Factors That Increase Potential for Groundwater Contamination

- shallow groundwater
- rainy climate
- sandy soil, low organic matter
- soluble chemicals that are not adsorbed well onto soil particles
- bare soils without rhizosphere microbial populations
- chemicals that are injected or watered into soil

CONTACT OR PROTECTANT FUNGICIDES

Fungicides are categorized by how they interact with plant tissue after application. Some remain on plant surfaces, where they function as a **contact fungicide** and are sometimes called "protectants." They tend to be effective against a broad range of fungi. They do not promote the development of resistant populations of fungi because their modes of action target multiple metabolic sites. In periods of rapid plant growth or rainy weather, they must be reapplied every 5–10 days. Complete plant coverage is necessary during application.

Contact or Protectant Fungicides

- must be on plant surface before infection
- broad-spectrum activity
- resistance problems unlikely
- repeated applications required as plant grows or rain dilutes the chemical

Examples of Commonly Used Contact/Protectant Fungicides

- captan
- chlorothalonil (e.g., Bravo, Daconil)
- ethylene-bis-dithiocarbamates (EBDCs) (e.g., mancozeb, maneb)
- fludioxonil (e.g., Maxim, Medallion)

SYSTEMIC OR PENETRANT FUNGICIDES

These fungicides are absorbed (penetrate) into plant tissues. Because they kill or inhibit fungi without harming the plant, their modes of action tend to be quite specific, sometimes inhibiting a single enzyme system in a fungus. As a result, fungicide resistance is more likely to occur with repeated use. Systemic fungicides also are effective for a more-limited number of fungal diseases. Because they are absorbed, they do not wash off a plant and therefore require fewer applications. Despite the name “fungicide,” some chemicals are really fungistats, which inhibit growth but do not actually kill the fungus.

Locally systemic fungicides enter plant tissue but do not move far from the point of penetration. Other systemic fungicides, the **xylem-mobile systemics**, move upward in the xylem after they are absorbed and can move into new tissue as the plant grows. Only fosetyl-Al and other phosphites are **amphimobile** or **phloem-mobile systemics** and move upward, as well as downward into the roots after application.

Calibration of application equipment is very important when using systemic fungicides because **phytotoxicity** may occur if applications are too concentrated. Sometimes systemic fungicides are applied for **curative uses**, i.e., after infection has already occurred. Although systemic fungicides are absorbed into plant tissues and have some curative action, this is usually limited to the very early stages of infection, and the fungicides cannot be used to cure a severely diseased plant. Systemic fungicides cannot bring dead tissue or dead plants back to life. However, systemic fungicides make the use of environmentally based predictions of **infection period**s more effective and offer alternatives to routine calendar-based spraying.

Systemic or Penetrant Fungicides

  - **locally systemic**
  - **xylem-mobile** (moving upward in the plant)
  - **amphimobile** (phloem-mobile, moving both upward and downward in the plant)

- do not wash off
- fewer applications needed
- some limited curative (after-infection) activity
- more efective with disease predictions because of after-infection activity
- may be phytotoxic if too concentrated

- narrower spectrum of disease control
- resistance may develop with repeated use

Examples of Systemic Fungicide Groups

- **benzimidazoles:** xylem-mobile (upward movement) Example: thiophanate-methyl (Cleary's 3336, Topsin)
- **dicarboximides:** locally systemic Example: iprodione (Chipco 26GT, Rovral)
- **phosphites:** phloem-mobile Example: fosetyl-Al (Aliette)
- **Q_oI, strobilurins:** xylem-mobile Example: azoxystrobin (Abound, Heritage, Quadris)
- **sterol inhibitors** (DMIs, SBIs): xylem-mobile Example: propiconazole (Banner, Tilt)

RESISTANCE MANAGEMENT

Contact fungicides have multisite modes of action (i.e., multiple ways to attack fungal activities), so fungi are unlikely to become resistant to them. Some contact fungicides have been used for more than 40 years without resistance problems. Systemic fungicides usually have a single-site mode of action, so fungi are more likely to become resistant to them. Resistant strains or races of a pathogen are usually already present when a fungicide is first used. Continued use of the fungicide selects for survival of the resistant strains. Good stewardship of valuable fungicides reduces the chances that resistance of a pathogen to the fungicide will occur. Therefore, using fungicides only when needed and as part of an integrated program is recommended. There are some additional factors to consider in fungicide stewardship.

It is important to know what **active ingredient** or ingredients are in a fungicide because repeated use of the same active ingredient or the same **chemical group** of fungicides can lead to resistance. All commercial fungicides have brand or trade names that are used in commerce, but the chemical name of any active ingredient also must be listed on the label. Some active ingredients are sold under several different trade names, so applicators might believe that they are using different active ingredients when they are not.

Fungicides for Oomycetes

Oomycetes (*Pythium, Phytophthora*, downy mildew and white rust pathogens) are not true fungi. Several fungicides are effective only against oomycetes, but others often are not effective for diseases caused by oomycetes. Some of the newer Q_oI/strobilurins are unusual in that they do have some activity against oomycetes as well as against diseases caused by true fungi. Fosetyl-Al is mostly used for oomycete diseases, but has some activity against other fungi.

Oomycete fungicides

- etridiazole
- fosetyl-Al
- mefenoxam/metalaxyl
- propamocarb
- zoxamide

It also is necessary to determine the chemical group or family to which each fungicide belongs because all chemicals in the group have the same mode of action. Even though an applicator might apply different active ingredients, the pathogens will react in the same way if the chemicals all have the same mode of action.

Whenever a new fungicide becomes available, it is important to ask for the name of the active ingredient and its mode–of-action group. The Fungicide Resistance Action Committee (FRAC) has established FRAC codes that indicate which active ingredients have the same mode of action. It is not necessary to memorize this information, but all applicators should have access to the trade names of the products they use, their active ingredients, and their FRAC codes. This information is available from the FRAC website, chemical sales representatives, manufacturer websites, or university extension specialists.

How to prevent or delay the development of resistance is a matter of some controversy, but the repeated use of a single fungicide or fungicides from the same chemical group, i.e., with the same mode of action, will certainly lead to resistance more quickly. Some chemical groups are at a higher risk for resistance because a single gene in the pathogen confers resistance. Other chemical groups are less likely to result in resistance because several genes in the pathogen are involved in resistance.

To Reduce the Chances of Fungicide Resistance

1. Minimize conditions favorable for disease development.
    - Use good cultural practices.
    - Use resistant cultivars if available.
2. Make good fungicide choices.
    - Be sure of your diagnosis.
    - Decide if the problem warrants a treatment.
    - Read the label.
    - Calibrate and apply properly.
3. Know the chemical.
    - What is the active ingredient?
    - Is it a contact or a systemic fungicide?
    - If fungal resistance is a concern, what is its chemical group?

- Change groups. Mix or alternate fungicide groups or include a contact fungicide that is not subject to resistance problems.

Another factor that affects resistance management is how the fungicide-resistant strains of a pathogen interact with fungicide-sensitive pathogen strains. Sometimes, resistant strains of the pathogen are competitive and persist in the environment even after the fungicide group is no longer used. If the resistant strains are less competitive than the sensitive strains, withdrawing that chemical group from use for some time might allow the fungal population to return to having sensitivity to that chemical group.

Most scientists believe that it is best to try to prevent or delay resistance rather than to manage it after it occurs. They recommend that systemic fungicides from a chemical group with the same mode of action be **mixed** or **alternated** with a systemic fungicide with a **different** mode of action or with a contact fungicide that is not subject to resistance problems. Applications based on infection-period forecasts may reduce the number of fungicide applications used compared to the number required by calendar-based schedules. Fewer applications mean less selection pressure on the pathogen to become resistant to the fungicide.

> “There is no beginning to love,” Roger said. “It just creeps over you.”
>
> “Oh,” said Hilma, “like brown rot on a plum tree in the dark winter months, and by the time you become aware of it, the leaves are out and it’s too late to spray.”
>
> —Bailey White, *Quite a Year for Plums* (Random House, New York, 1999)

Chemicals play an important role in the protection of plants against many foliar diseases. All pesticides should be used with care to avoid environmental contamination, phytotoxicity, residues in food, and applicator exposure. Plants are sometimes protected against bacterial infections with antibiotics or copper-containing compounds. Most fungicides used today are organic compounds with low mammalian toxicity (i.e., high LD_{50}) that are degraded by bacteria. Contact or protectant fungicides remain on the leaf surface, are effective for a wide range of fungal diseases, are not generally at risk for development of resistance, and must be reapplied after rain or plant growth. Systemic or penetrant fungicides may be absorbed locally, move upward in the xylem, or be amphimobile in the phloem. They do not wash off, are effective against fewer diseases, and may have some after-infection activity. Because systemic fungicides have more specific modes of action, they are at risk for development of fungal resistance. Systemic fungicides should be rotated or mixed with systemic fungicides having a different mode of action or with a contact fungicide that is not subject to resistance problems. The development of resistance can be minimized by using fungicides only when needed and as part of a resistance management program.

Biological Control

Evidence of Natural Biological Control

- the sporadic nature of disease
- efectiveness of crop rotation
- suppressive soils

- disease declines
- increase of a disease when a fungicide is applied for treatment of a diferent disease
- increase in disease severity following introduction of a pathogen to sterile soil compared to soil with natural microbial populations

As discussed earlier in this chapter, there is good evidence that natural biological control is at work in agroecosystems. The sporadic nature of disease suggests that pathogens are kept at bay unless conditions are particularly favorable for infection. With the use of fungicides that target only certain pathogens, we sometimes see an increase in a certain disease when a fungicide has been applied to manage a different disease. This may be due to the impact of the fungicide on natural competitors and antagonists that usually inhibit the nonmanaged pathogen. Increased disease severity also has been observed following the introduction of pathogens into sterile potting media or in sand-based putting greens with little organic matter to support microbial activity. These observations have encouraged plant pathologists to try to isolate specific organisms that could be applied to plants to protect them against infections. The modes of action of biological control agents used to protect plants from infection include antibiosis, competition, and cross-protection.

ANTIBIOSIS

It is difficult to separate competition and antibiosis because many antagonists use both methods. However, some specific antibiotics are produced by antagonists of plant pathogens (Figures 11.22B and 11.33). Several fluorescent *Pseudomonas* bacteria found in the rhizosphere produce antibiotics and play a significant role in disease decline phenomena (Figure 11.34). A commercial biological control, Nogall, contains *Agrobacterium radiobacter* strain 1026, which produces **bacteriocins**—antibiotics that specifically inhibit the crown gall bacterium in many host plants. Studies have shown that competition at potential infection sites also plays a role in the success of this product.

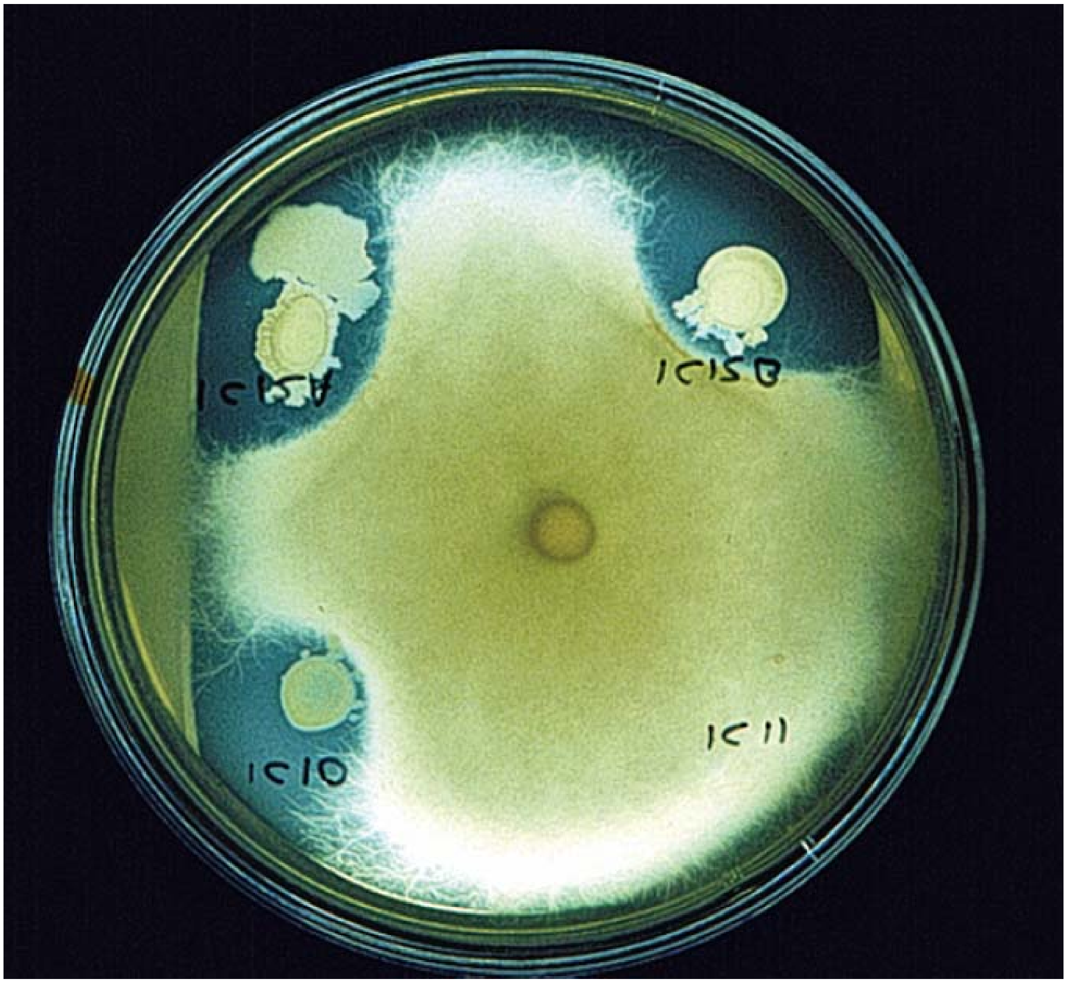


Figure 11.33. Inhibition of *Gaeumannomyces graminis*, the fungus causing take-all root disease, by three different bacteria. The bacteria produce antibiotics that inhibit the growth of the fungus.

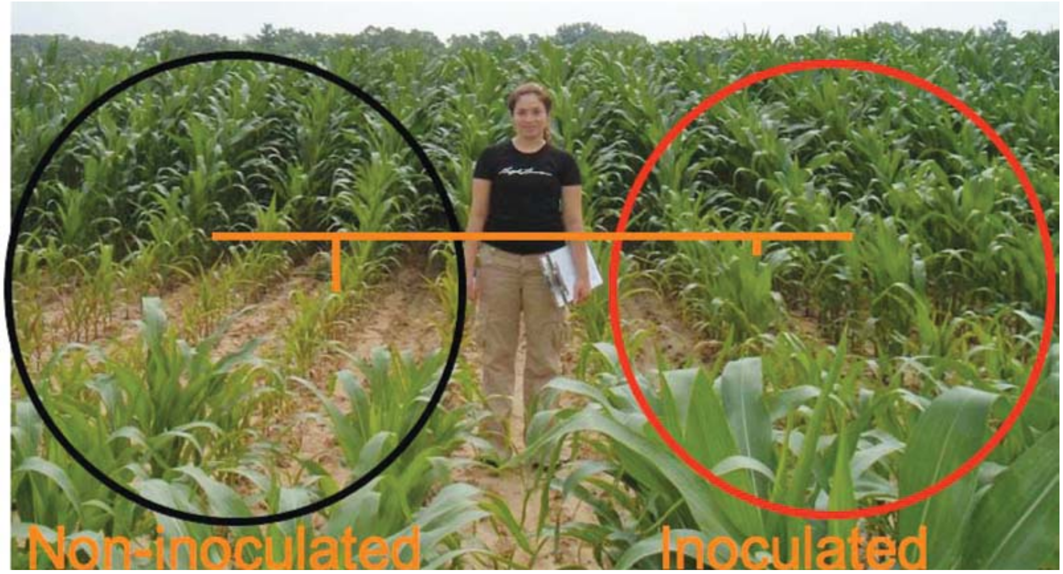


Figure 11.34. Use of seed inoculation with rhizosphere-competent strains of *Pseudomonas fluorescens* under acidic soil conditions to improve corn growth in the field.

COMPETITION

Microorganisms compete for food and space on plant surfaces. In many cases, timing is an important factor. In nature, it is often “first come, first served.” This can be exploited by applying competitive organisms on leaf and root surfaces to reduce infections by pathogens. A successful example is the use of a competitor to prevent a wood decay basidiomycete, *Heterobasidium annosum,* from colonizing freshly cut tree stumps during timber harvesting. This decay fungus can spread from stumps to neighboring, healthy trees through root contact. A competitive, saprophytic basidiomycete *Phlebia gigantea* (syn. *Peniophora gigantea*) was isolated from old stumps, and basidiospores were then applied to stumps immediately after cutting to prevent colonization by the wood decay fungus. The bacterium *Pseudomonas fluorescens* strain A506 is registered and sold commercially (as BlightBan A506) for protection of apple trees against fire blight. Its mode of action includes both antibiosis and competition.

CROSS-PROTECTION

The phenomenon of cross-protection was discussed in Chapter 9. Cross-protection uses mild virus strains to protect against infection by severe strains. This method has largely been replaced by the use of genetic engineering of plants by inserting viral coat-protein genes and other viral genes that protect plants from infection without the problems associated with infections by mild strains of viruses.

SUCCESSFUL USE OF BIOLOGICAL CONTROL

The successful use of a biological control agent for plant protection depends on the following factors.

1. **Source of the organism.** Organisms isolated from plant surfaces are most likely to be successful because they are adapted to the environment in which they must be effective. Such organisms are described as **phylloplane-competent** or **rhizosphere-competent.**

2. **Proper application of specific isolates.** It is unlikely that artificially high populations of applied agents will be maintained for very long. The natural microbial interactions will result in more balanced populations fairly quickly. The use of disease prediction systems might help by determining the specific times when protection is needed. At the very least, it is likely that repeated applications of biological control agents will be necessary to maintain the high populations necessary for efficacy. Even though there are many reports that certain species or genera of fungi and bacteria are likely to function as competitors and antagonists, isolates vary considerably in their impact on pathogens. Labels of biological control products should indicate which specific isolates are provided and not just list species or genus names of possible agents (Figures 11.35 and 11.36).

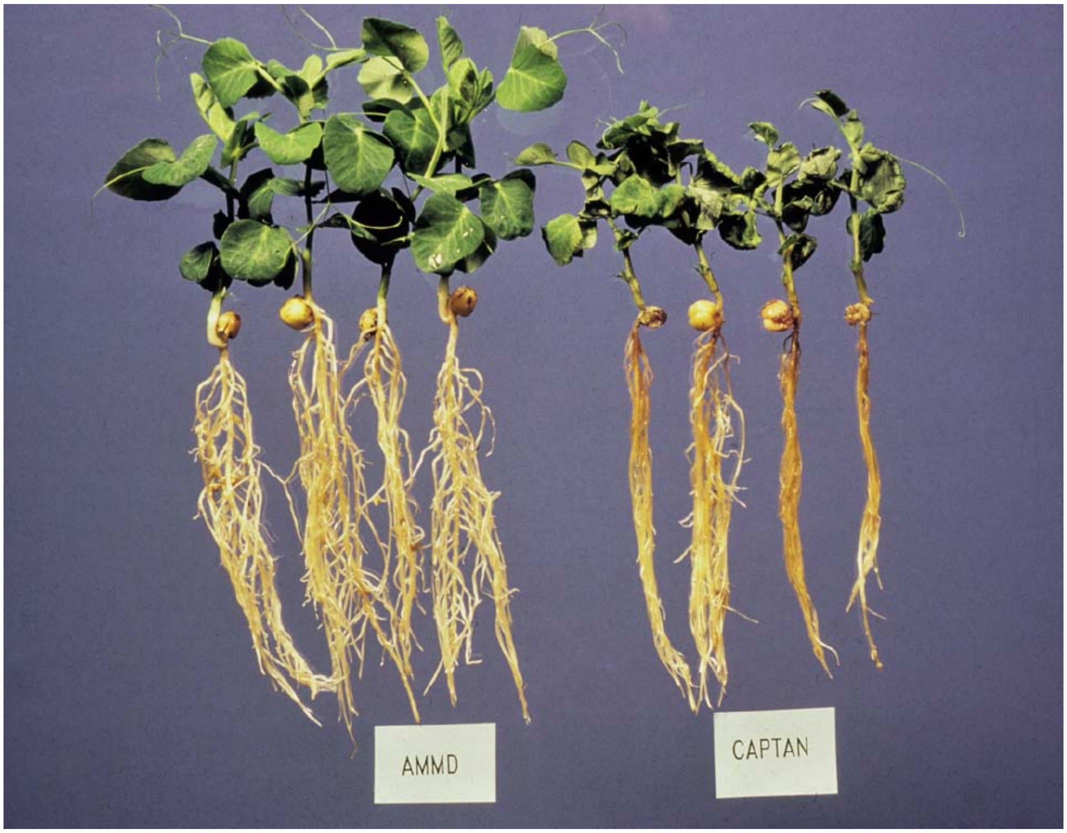


Figure 11.35. Biological control of Aphanomyces root rot of pea. Left, pea seeds coated with the bacterium *Burkholderia cepacia* (*Pseudomonas cepacia*) strain AMMD. Right, pea seeds treated with the fungicide captan. All plants were inoculated with zoospores of the pathogen.

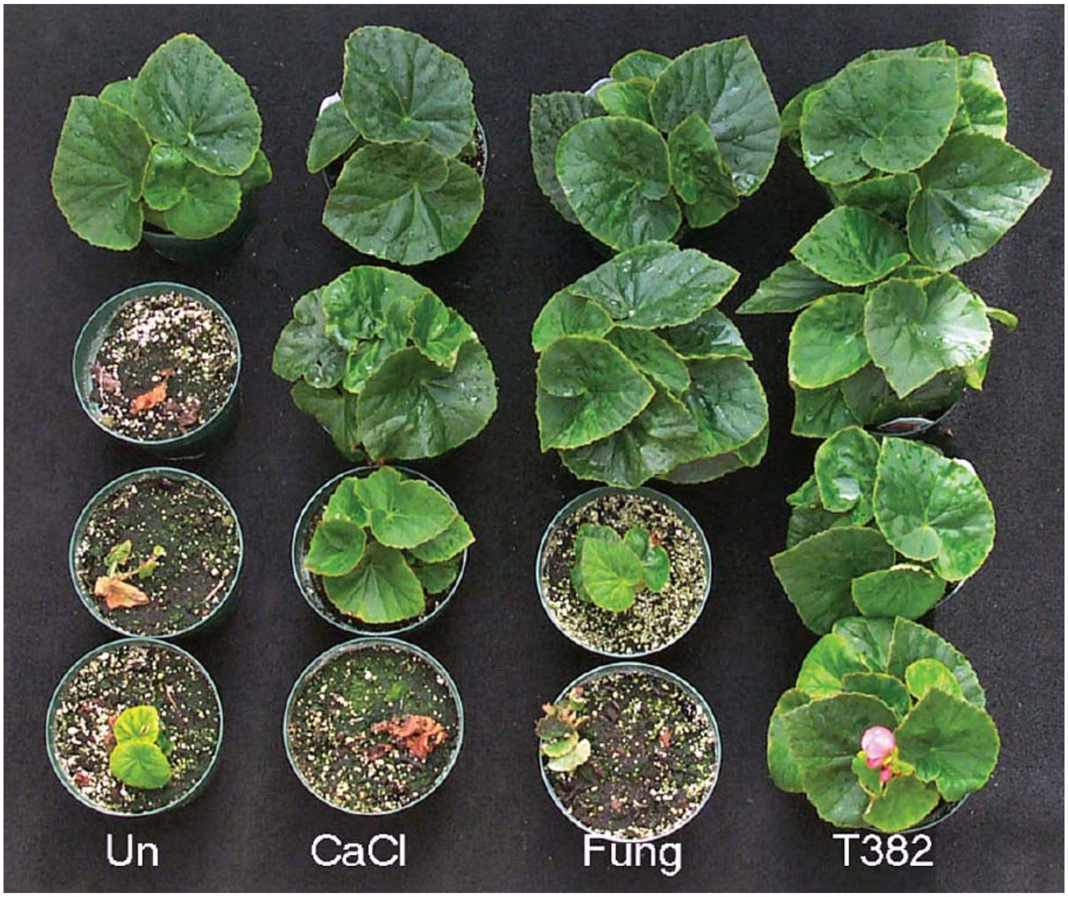


Figure 11.36. Begonias grown in the greenhouse and inoculated with *Botrytis cinerea*, the fungus causing gray mold, under conditions optimal for the development of disease. Treatments (with four replications) from left to right: untreated (Un), $CaCl_2$, chlorothalonil fungicide (Fung), and a biological control fungus *Trichoderma hamatum* T382 inoculated into the potting mix (T382).

3. Formulations. Formulations must allow biological control organisms to be stored in a convenient form and become active quickly using a variety of water supplies, including chlorinated municipal water. Spores and freeze-dried fungal preparations are amenable to this kind of packaging. Some bacteria such as *Bacillus* species produce thick-walled endospores that have made them popular choices because they are easily stored in dry formulations. *Pseudomonas* bacteria, which do not produce endospores, may be formulated in liquid media that require refrigeration.

4. Safe handling. Even though many people would like to use biological control products as an alternative to more toxic chemicals, it should be remembered that concentrated populations of microorganisms have their own potential risks. Inhaled spores of fungi can trigger allergic responses. Some soil bacteria might be secondary human pathogens and threaten the health of people with compromised immune systems. Safety instructions on labels should be followed.

5. Reliable information. Many biological control products are promoted on the basis of experiments in laboratories and greenhouses and only on certain kinds of plants. Evaluations by independent observers should be consulted to determine whether a product is successful in field conditions, in the environment in which it will be used, and on the plants to which it will be applied. Many biological controls are effective against specific diseases only. The Office of Pesticide Programs of the U.S. EPA maintains a website that lists biopesticide fact sheets that describe each agent, modes of action and applications, and risks to human health and the environment (Figure 11.37).

Things to Consider Before Buying a Commercial Biological Control Product

1. Have independent researchers with no financial interest in the product published evaluations of the product?
2. Has the product ever been tested on the plants for which you wish to use it? For which diseases is it recommended?
3. Has the product been tested in your area?
4. If no independent data are available, look for this information:
    - Nontreated plots as controls for comparison with the treatments.

  - Replications of the treatments, not just a single area of application.
  - Statistical analysis of the data.
  - EPA registration. Some companies evade regulations for safety by claiming that the product makes plants less susceptible to disease.

5. Ask for a free sample to do your own evaluation: Try new products on a small area first where they are least likely to cause problems if they don't work. Leave a nontreated area for comparison.

got aflatoxin?
get
afla-guard
CIRCLE ONE GLOBAL
SoilGard 12G
Contans®WG
N-Hibit™
seed treatment
MYCOSTOP
BIOFUNGICIDE
KEEP OUT OF REACH OF CHILDREN
CAUTION
Verdera
SERENADE MAX
VAULT™
A LEAP AHEAD
PlantShield HC
BioWorks, Inc.
BioStacked
Ballad PLUS
BIOFUNGICIDE
Now Available!

Figure 11.37. Some examples of commercial biological control products (biopesticides).

Some Organisms Found in Several Commercial Biological-Control Products Used to Protect Plants from Infection

Bacteria

- *Bacillus subtilis*

- *Burkholderia cepacia* (syn. *Pseudomonas cepacia*)
- *Pseudomonas fluorescens*
- *Pseudomonas syringae*

Fungus

- *Trichoderma harzianum*

The modes of action that are most useful in protecting plants using biological control agents are competition and antibiosis. Specific isolates of fungi and bacteria from plant surfaces are most likely to be successful, but repeated applications will probably be necessary. Research to develop products that will work reliably in field situations is ongoing.

Genetic Resistance

Genetic resistance, discussed in Chapter 9, is usually available as specific (R gene, vertical) resistance that works against specific races (or strains) of a pathogen or as general (horizontal) resistance that usually is partially effective against all races of a pathogen (Figures 11.38–11.40). **Specific resistance** is commonly used for monocyclic pathogens. The probability of selecting races of a pathogen that can overcome the resistance is reduced because the pathogen completes only one generation per season. Many monocyclic pathogens are soilborne, so even if such races are selected, they are less likely to be quickly and widely distributed. Some R genes have remained effective for many years. Specific resistance can be considered a means of reducing primary inoculum because it is effective for specific races of the pathogen.

Figure 11.38. Elm seedlings in the greenhouse prior to testing for resistance to Dutch elm disease.

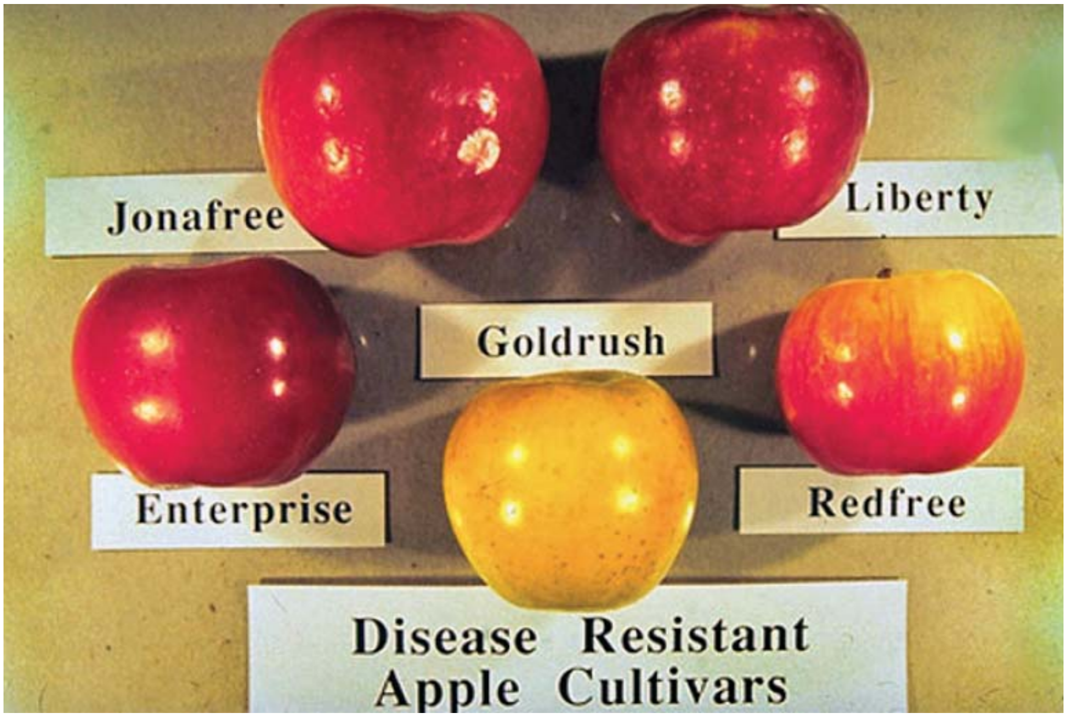

Jonafree
Liberty
Goldrush
Enterprise
Redfree
Disease Resistant
Apple Cultivars

Figure 11.39. Some apple cultivars that are resistant to apple scab.

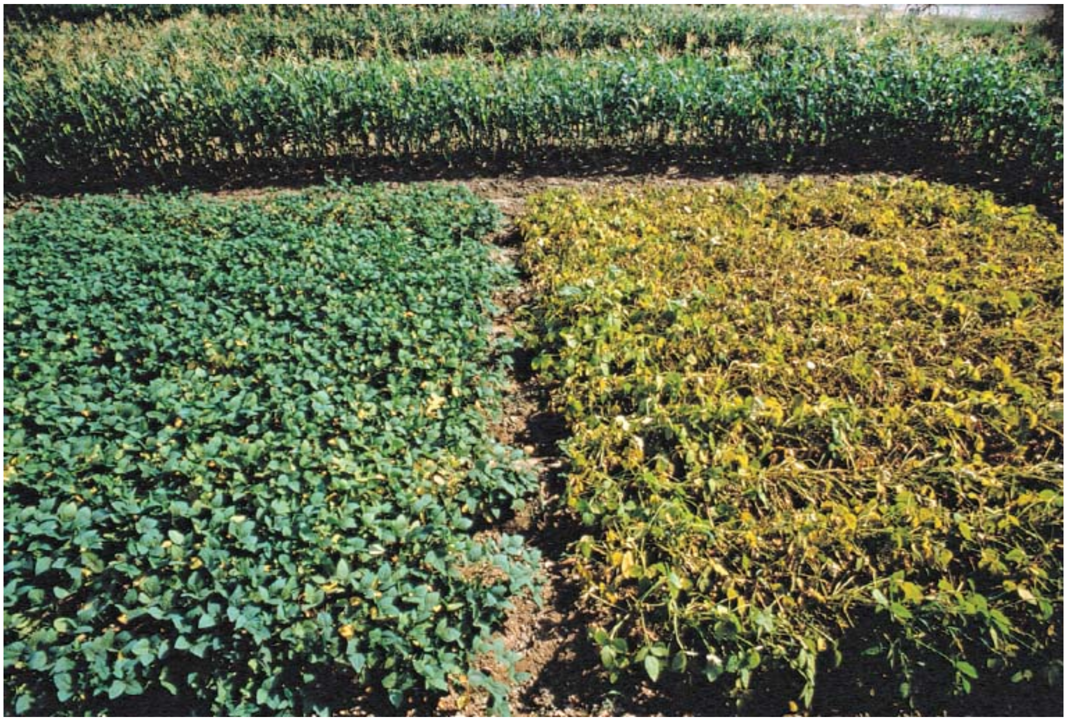


Figure 11.40. Experimental plots with plants tolerant (left) and susceptible (right) to soybean rust.

Genetic Resistance for Monocyclic Pathogens

Specific (R gene) resistance is often effective when one strain or race of a pathogen dominates the population. This reduces primary inoculum because the resistance gene is effective against a single race of the pathogen.

To Prolong the Usefulness of the Specific Resistance Gene(s)

Use all appropriate cultural practices to reduce the population of the pathogen, e.g., clean up infested debris, rotate crops, minimize leaf wetness for foliar pathogens, and use general resistance.

General resistance is a form of resistance that reduces the rate of an epidemic. Lesions are usually smaller, develop more slowly, and produce less secondary inoculum on plants with general resistance. This slows the rate of disease increase throughout the growing season and is therefore very useful for polycyclic pathogens. In seed catalogs, this type of resistance is often called “tolerance,” to warn people that the disease might still occur but it should be less severe on that cultivar. (Note from Chapter 9 that plant pathologists use the word *tolerance* differently.) General resistance is what allows plants to grow in the presence of a number of minor pathogens without significant economic impact. Environmental conditions and cultural practices affect how well or how poorly general resistance performs.

General resistance may not be sufficient to manage disease at a desirable level and is not easy to select quickly in breeding programs. Can specific (R gene) resistance be used for polycyclic pathogens without the danger of the “boom-and-bust” phenomenon? The methods discussed below have been developed to use R genes successfully against polycyclic pathogens. Many of these are used in cereal crops.

Genetic Resistance for Polycyclic Pathogens

General resistance reduces the rate of the epidemic. It is efective against all races of a pathogen. Lesions are smaller, take longer to form, and produce less inoculum. This may or may not be sufficient to meet economic thresholds.

General resistance can be enhanced by good cultural practices that reduce the pathogen population and its opportunities to infect the plant.

Use of R-gene resistance for polycyclic pathogens

- geographic deployment
- temporal deployment

- gene pyramiding
- multilines or mixtures

► DID YOU KNOW?

Seventy-five percent of crops grown in the United States are resistant cultivars. This rises to 95–98% when small grains and alfalfa are considered.

GEOGRAPHIC DEPLOYMENT

Although this requires good cooperation among seed suppliers and farmers, different R genes may be used in different regions. As spores arrive from different areas, they will meet new R genes and be unable to infect the plants.

TEMPORAL DEPLOYMENT

By monitoring the races of widespread, airborne pathogens, seed producers can provide different R genes in different years to help protect plants from the current year's inoculum.

PYRAMIDING

In some cases, plant lines with different R genes are crossed repeatedly until multiple R genes are present in a single cultivar. It is very unlikely that a pathogen would overcome all of the R genes. On the other hand, the possibility of selecting a "super-race" that could overcome all of the genes at once makes some scientists somewhat fearful that the usefulness of all of the R genes could be lost.

MULTILINES AND MIXTURES

Multilines and mixtures are alternatives to pyramiding. **Multilines** are a set of inbred cultivars that vary only in the R gene that they carry. These cultivars are physically mixed together and planted to create a plant population similar to what might be found in a natural ecosystem. Multilines have been used successfully to manage stripe rust in the U.S. Pacific Northwest. **Mixtures** of lines that are not so similar are sometimes used in a parallel way. Each plant has only a single R gene, but its close neighbors will have different R genes. If an infection occurs, secondary inoculum is less likely to land on a susceptible plant. Multilines and mixtures offer flexibility because breeders can add or subtract R genes each year based on pathogen monitoring during the previous season. Some argue that multilines and mixtures also are less likely to select for a "super-race" of a pathogen that could infect all of the plants because of a phenomenon called **stabilizing selection**. It is theorized that excess virulence genes actually decrease pathogen fitness, so a super-race would be at a competitive disadvantage compared to simpler races in fields of multilines or mixtures and would, therefore, be less likely to occur (Figure 11.41).

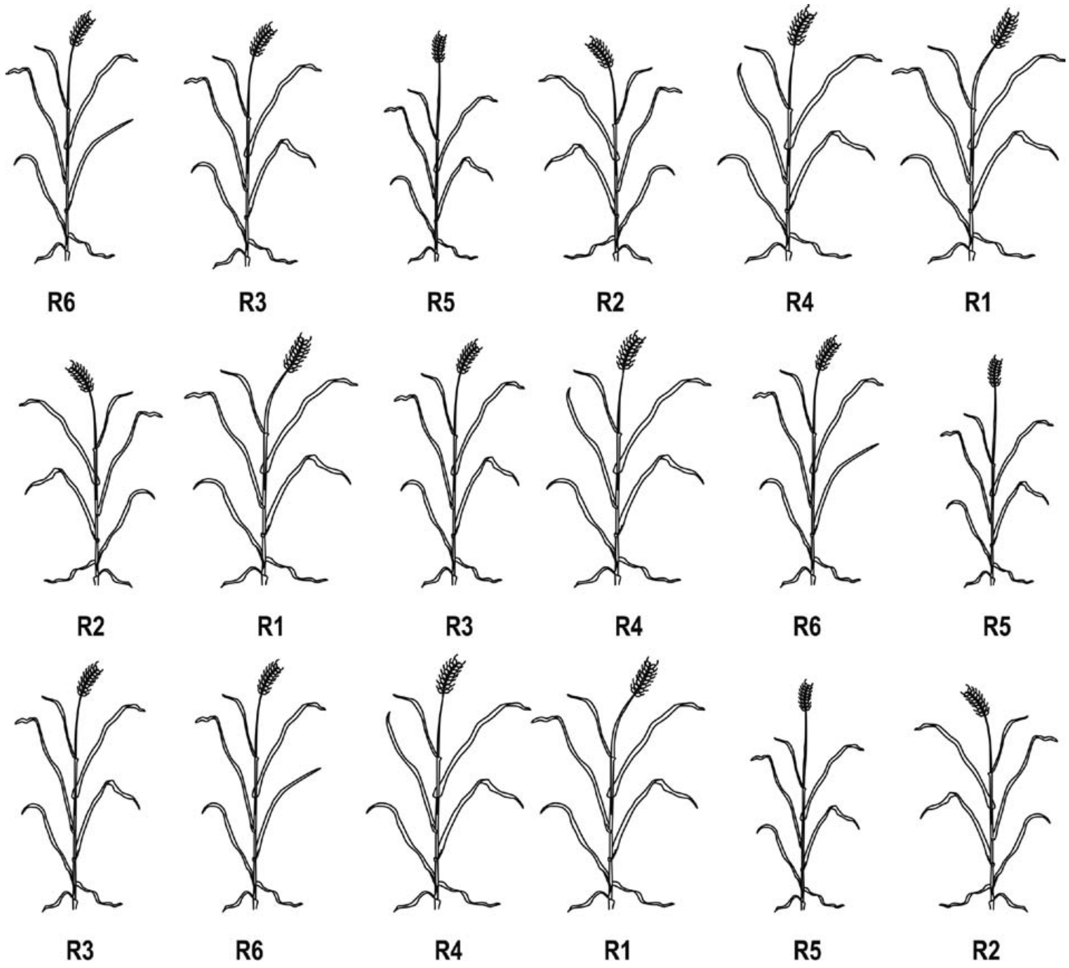


Figure 11.41. Diagram of multilines of a cereal crop showing a mixture of six R genes planted out randomly.

Advantages of Genetic Resistance

- Introduced gene is inherited and permanent.
- No action is needed on the part of the grower.
- Resistance genes are likely to function in a new genetic background after crossing.
- Resistance does not harm the environment.
- Resistance is compatible with other disease management methods.

Biotechnology, including **genetic engineering**, offers new ways to create plants with genetic resistance. Currently, the most common genetically engineered plants are those with resistance to a nonselective herbicide or with insect resistance. Insect resistance is primarily conferred by genes from the insect-pathogenic bacterium *Bacillus thuringiensis* (*Bt* genes) that produce toxins that poison certain insects. Virus resistance from coat-protein genes and other viral genes is the most common disease resistance product at this time (Figure 11.42). Virus-resistant papaya, potato, squash, and tomato are available through genetic engineering. Researchers have identified and cloned a number of resistance genes and have developed novel approaches to interfere with pathogen infection. These provide opportunities to reduce the use of fungicides and provide durable resistance to the major diseases.

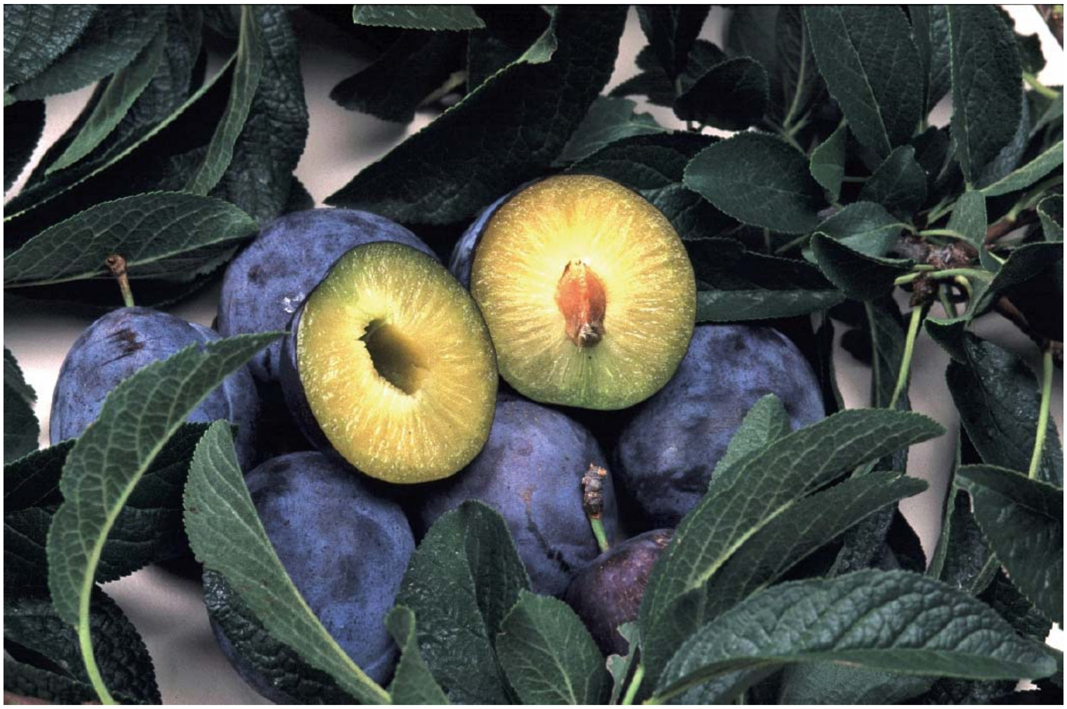


Figure 11.42. Resistance to *Plum pox virus* in transgenic plum clone C5 due to the insertion of the PPV coat protein gene into the plant genome.

Every year, plant pathologists publish the results of evaluations of resistant cultivars, biological agents, and cultural practices for plant disease management in the online journal *Plant Disease Management Reports*. Crop-based organizations also publish cultivar resistance evaluations and make them available to their members and through local university extension programs.

Biotechnology and Genetic Engineering

Limitations to producing genetic resistance

- Single genetic factors may be widely distributed in crop plants (genetic uniformity).
- Eukaryotic genes and systems are complex, compared to those of prokaryotes.
- Many characteristics are not governed by a single gene.
- Gene transfer by genetic engineering is not an efficient process.

Advantages of producing genetic resistance

- Introduced resistance genes may be available more quickly in a background of acceptable agronomic characteristics (multiple generations of backcrossing not needed).
- Resistance genes from genetically isolated organisms become available.
- Genes may be introduced into reproductively sterile plants.
- Resistance genes could be deployed as needed or in variable combinations more quickly.
- In the future, it may be possible to create synthetic resistance genes.

Genetic resistance is an important means of plant protection. Specific (R) gene resistance is used for monocyclic pathogens and in some specialized ways for polycyclic pathogens. It is a means of reducing primary inoculum. To avoid the "boom-

and-bust" cycle, it is important to minimize the selection pressure on the pathogen to overcome specific resistance. General resistance is partial and reduces the rate of disease increase. It is used for many polycyclic pathogens and can be modified (strengthened or weakened) by environmental conditions and cultural practices. Biotechnology and genetic engineering offer new means of creating resistant plants.

How can we create integrated, sustainable disease management programs?

Constraints to plant production vary depending on the crop- or plant-production system. All plants have an absolute genetic potential that could be reached if there were no limiting factors (Figure 11.43). This "absolute" level of productivity cannot be reached in commercial plant production. Ideal yields and optimal growth may be reduced by physical and chemical extremes in the environment, including extremes of temperature, soil pH, or rainfall. These vary from year to year and from area to area. The environment determines the "attainable" level of production. The "affordable" level of production is affected by the economics of plant production—the short-term costs to the grower and the long-term costs to the industry and eventually to society. Short-term costs include irrigation water, fertilizer inputs, and chemical and biological control applications. Long-term costs include environmental pollution, loss of biodiversity, investments in new production and harvesting equipment, land rotation schemes, storage facilities, and water supplies. Once management decisions have been made, and the plants are growing, unexpected factors might reduce production even more. Hazards such as diseases, drought, frost, hail, or pests will determine the "actual" level of production in any given year.

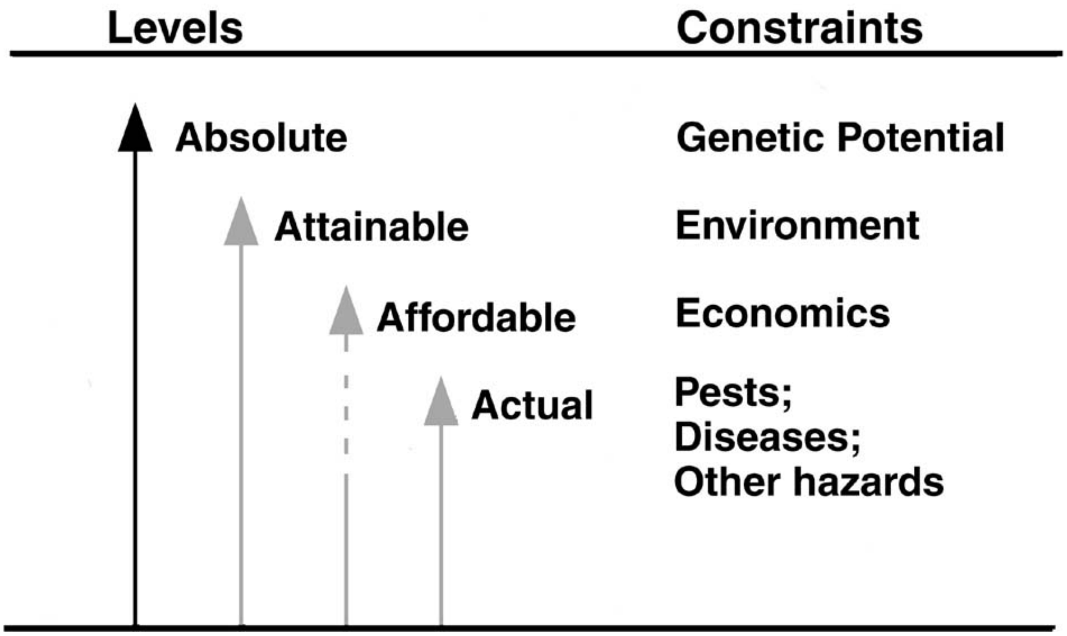

Levels
Constraints
Absolute
Genetic Potential
Attainable
Environment
Affordable
Economics
Actual
Pests;
Diseases;
Other hazards

Figure 11.43. Constraints to crop production. See discussion in text.

► **DID YOU KNOW?**

In the United States, only 2–3% of the population works directly in agriculture. Food costs, on average, make up about 15% of individual disposable income.

Disease management strategies are an important part of any crop management plan. Management of a plant disease usually includes multiple methods. If the pathogen is monocyclic, the focus will be on reducing the amount or efficacy of the primary inoculum. If the pathogen is polycyclic, the relative amount of focus on reducing primary inoculum or on reducing the rate of disease increase depends on the specific disease, but usually methods that accomplish both are recommended.

“**Integrated pest management (IPM)** is a site-specific, multitactic decision-making process for the management of a pest that is profitable for the grower, and promotes health and environmental quality.”

—Tree Fruit IPM Working Group, Washington State Horticultural Association

It is not difficult to understand that relying on a single management method is risky, but it is not simply a matter of the potential success or failure of that single method. Many methods interact with each other synergistically. For example, a good cultural program of crop rotation, removal of infested debris, moisture management, and care in application of fertilizers reduces the pathogen population and reduces opportunities for infection. When resistance genes are used, the probability of selecting a pathogen strain that can overcome the resistance is reduced by the cultural program. Many agricultural crops are protected from diseases using only cultural practices and resistant cultivars. Home gardens often can be very productive without any pesticide use.

When fungicides are necessary, it is less likely that fungicide resistance problems will arise if the pathogen population is reduced and infection opportunities are minimized. Cultural practices can reduce the need for fungicides, especially when weather does not favor infection. Further reductions in fungicide use can be made when a good cultural program is combined with environmental monitoring of infection periods. Applications may be skipped entirely in dry weather, or the interval between sprays may be extended.

► DID YOU KNOW?

Norman Borlaug won the Nobel Peace Prize in 1970 for his efforts in wheat breeding because the Nobel

> committee recognized the importance of food supply to political stability.

When we introduce biological controls and systemic acquired resistance (SAR) into management programs using plant resistance and pesticides, pathogens are less likely to overcome resistance genes in the plant or become resistant to pesticides. SAR is a protective biological reaction that is affected by the health of the plant and its ability to quickly produce defense compounds (see Chapter 9). Genetic resistance also relies on the defense reactions made by the plant when it is invaded. The cultural practices used to create an optimal environment for plant growth and development favor a rapid and effective biological defense response.

We do not have an unlimited supply of chemicals that can serve as plant protectants or an unlimited source of resistance genes in the gene pool of the world's organisms. The stewardship of these valuable tools in the fight against plant diseases lies in the hands of those who use them. We have a responsibility to use them wisely and with minimal impact on the environment. Disease management programs that integrate cultural, chemical, genetic, and biological approaches are more likely to be successful and sustainable (Figures 11.44 and 11.45).

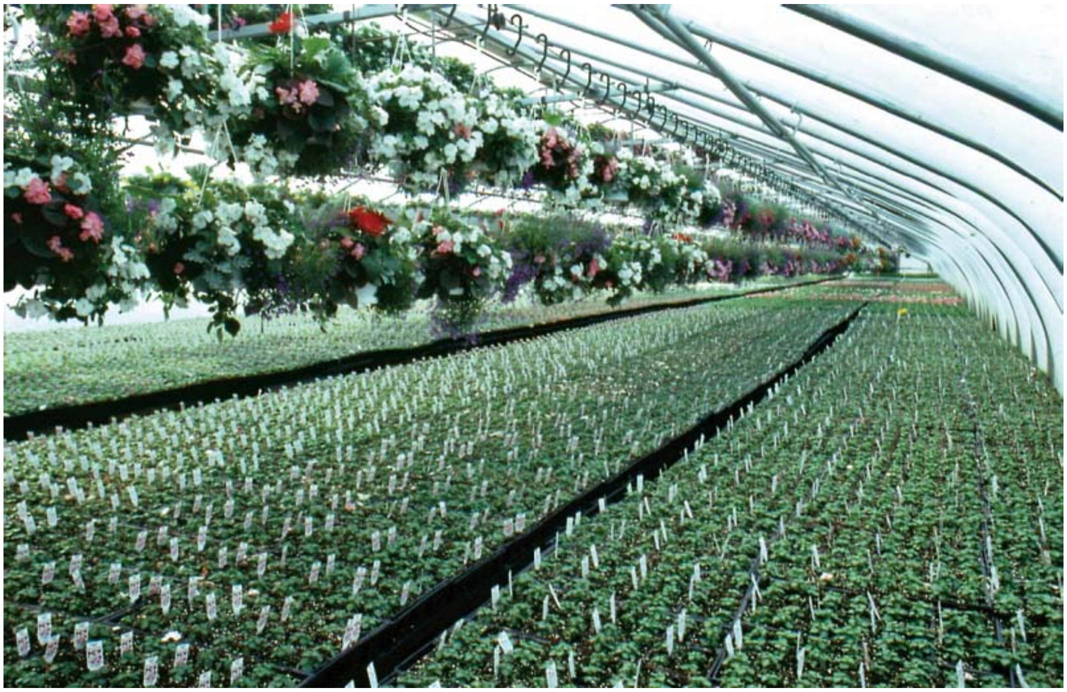


Figure 11.44. Modern agriculture puts plants at risk with genetic uniformity, as illustrated by this greenhouse full of genetically uniform geraniums.

► DID YOU KNOW?

A measure of our world food security and the ability to meet the food demands of the world's population is the world grain stocks. The grain stocks are the amount of food grains stored around the world. In April 2004, world cereal grain stocks had fallen to their lowest level in 30 years, with only enough stored grain to supply the world for 59 days. Thus, the diseases that affect cereal crops become especially important when so little grain is in storage.

Figure 11.45. Rice culture in the highlands of the Philippines. Such intensive agriculture can increase the risk of plant diseases.

Putting it all together for sustainable crop management is a challenge, but a sound cultural program will be effective against a wide range of potential pathogens. This permits a grower to focus on more-threatening pathogens or on those that have specific management requirements. Some plant systems are more challenging than others. There are fewer options for perennial systems than for annual plantings. Modern technologies of environmental monitoring and rapid pathogen identification, using PCR and immunoassays, move growers from a “spray and pray” approach to one that is based on accurate diagnosis and environmentally sound management choices. Finally, our developing understanding of the details of the ecological, physiological, and genetic interactions of plants and their pathogens will result in novel disease management approaches for the future.

Providing food, fiber, and pleasing landscapes to a rapidly increasing world population challenges all plant managers. Plant diseases are an important factor in this struggle. Diverse management practices are more likely to be successful and sustainable. Diversity in the plants on which we rely also will improve the sustainability of our food (Figure 11.46) and fiber supplies. The biodiversity of the world is concentrated in the tropics, where it is most threatened by growing populations and urban development. Our individual choices in the use of energy and other resources, the environmental regulations we are willing to obey, and the support we give to future research and diagnostic facilities for plant health will determine the world of the future.

► DID YOU KNOW?

- Only 12 species of plants contribute significantly to the world’s food supply. Most of the calories

consumed by people on our planet are provided by only three species: wheat, rice, and corn.
- One-third of the world's food is grown on the 18% of the land that is irrigated.
- In the past 40 years, 30% of the world's soils have become unproductive, which has contributed to malnourishment of 3 billion people.
- It may take 500 years to make one inch of topsoil.
- There has been more population growth since 1950 than during the preceding 400 million years. There were 1 billion people in 1800 and 4 billion in 1975. There are nearly 7 billion today.

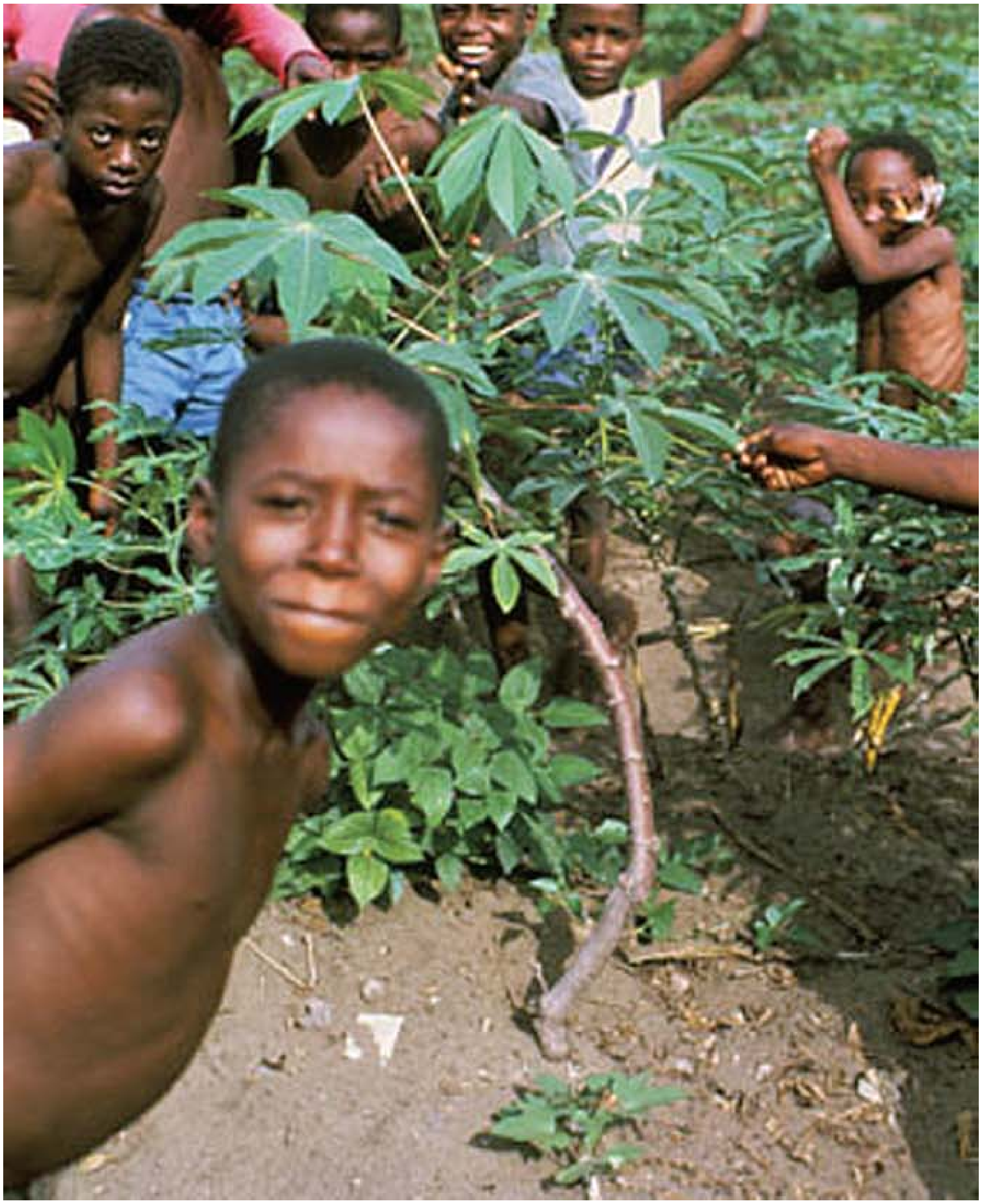


Fig. 11.46. A, Harvest of cassava (manioc) (***Manihot esculenta***) roots, an important crop in the tropics but not commonly consumed in temperate areas. B, African children harvesting cassava leaves. Crop diversity is important in providing a stable world food supply.

> “Everyone says something must be done—but this time it looks like it might be us.”

—Will Rogers

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American Phytopathological Society. Plant Health Management Series. APS, St. Paul, MN. The five books in this series explain strategies to achieve healthy tomato, potato, wheat, citrus, and peanut crops—from seed to storage. The books address the use of integrated pest management techniques against weeds, diseases, and insects. Information about the titles in this series is available at the APS website.

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Questions

General

1. What are the major approaches to plant disease management? Provide a brief definition for each.
2. Integrated pest management (IPM) is the mainstay of modern agriculture. How can IPM help maintain the effectiveness of disease resistance genes? How can IPM help reduce the chances of fungicide resistance problems? How can IPM help reduce the need for fungicides and make the needed application more efficacious?
3. Your town garden plots have a "no pesticide" policy, but the gardeners would like some guidance on disease management. What management practices would you recommend to

minimize plant diseases in this situation? Begin with preparation of the plots and choice of seed, continue with in-season activities, and finish with harvest and preparation for winter. Be sure to include activities from the major categories of management: avoidance, exclusion, eradication, and protection. Consider diseases caused by all of the major pathogen groups.

Avoidance

1. Why is avoidance of the pathogen of little practical value for most diseases?

Exclusion

1. What are some limitations to the use of quarantines to exclude pathogens?
2. What are some advantages to producing pathogen-free plants through meristem culture? What are some potential disadvantages in terms of disease management?

Eradication

1. What means of eradication could be used to reduce pathogen populations on plants of interest to you? How are the options different for annual outdoor plants, perennial outdoor plantings, and greenhouse crops?
2. What uses of heat could be applied to reduce pathogen populations in plants of interest to you?
3. What are some advantages and disadvantages of crop rotation, soil fumigation, and solarization for the eradication of soil pathogens?
4. Briefly define the three modes of action of biological control that are used primarily as methods of eradication.

Protection

1. How can leaf wetness be minimized in plants that you wish to grow?
2. What are the common modes of action for biological control used for protection of plants? What are some major limitations to the commercialization of biological control agents for plant protection?
3. What pesticide application regulations minimize residues in foods at the time of harvest? What precautions should applicators take to minimize their exposure? How can you minimize the risk of groundwater contamination by pesticides that you apply?
4. What general kinds of fungicides are available for plant diseases? The words “systemic” and “curative” are sometimes used to describe certain fungicides. How do fungicides move in plants to which they are applied? Can they cure fungal diseases?
5. What information about a pesticide should you obtain from its label before using it? What information should you know about a new fungicide product in order to develop a resistance management plan?
6. If specific (R gene) resistance is available for a monocyclic pathogen, why do you still need to use cultural practices and other management activities?
7. General resistance is most effective for polycyclic pathogens, but it is not always available or sufficient to protect the plant. How can R genes be used for polycyclic pathogens?
8. What are some advantages of genetic engineering over traditional breeding for the production of disease-resistant plants?
9. Plant activators are chemicals that are applied to plants to trigger systemic acquired resistance (SAR). Is this chemical control, biological control, or genetic resistance? Should plant

activators be allowed in organic crop production? Explain your answers.

Challenge Questions

General

1. You are the only plant pathologist on the first extraterrestrial colony. An epidemic is in progress on an indigenous crop. What do you need to know about the crop and the pathogen to develop a management plan for the disease?
2. What do you think are the five most important factors that will affect our ability to provide safe and abundant food and fiber to the world's population in the next 50 years?

Exclusion

1. You wish to establish quarantine regulations for the import of a new plant species. What information would you need to have about its pathogens and the diseases they cause in order to establish reasonable regulations?
2. What are some limitations (biological and political) to the successful enforcement of plant protection quarantines?
3. Choose a plant that is vegetatively propagated. What pathogens affecting this plant can be excluded through meristem culture?

Eradication

1. As the official "plant pathologist" for your family, which eradication methods do you recommend for your home gardens?

Protection

1. The continued registration of several fungicides is uncertain. Briefly, what is the role of fungicides in agriculture? What nonchemical alternatives exist? What would be the agricultural effects if all fungicides were banned?
2. Fungicide use is concentrated in the humid eastern half of the United States. Describe a plant production system in which:
    a. Cultural practices alone will produce an economically viable crop.
    b. Cultural practices must be supplemented with fungicides to produce an economically viable crop.

   ("Crop" may be interpreted broadly to include landscapes and turfgrass.) Explain why fungicides are needed or not needed in each case.

Supplementary Resources

Illustrated Glossary

Special Topics

Biological control of plant pathogens
Plant disease management
    植物病害管理的概念與原則 (Chinese)
What are fungicides?

Plant Disease Profiles

Black Sigatoka of bananas and plantains
Citrus canker
Coffee rust
Downy mildew of grape

Fire blight of apple and pear
Late blight of potato and tomato
Soybean cyst nematode disease
Stem rust of wheat
White pine blister rust

Laboratory Exercise

Synthesis and bioassay of a volatile fungicide

Case Studies

Ineffective fungicides: A case study on problems in selection and use of fungicides for disease management

Hosta takeover: A plant disease management case study

► WORDS TO KNOW

The words in this list are defined in the Illustrated Glossary.

alternate host
alternative host
amphimobile
antagonist
antibiosis
antibiotic
avoidance
bacteriocin
biological control
biotechnology
certification
compost

contact fungicide
crop rotation
cross-protection
days to harvest
disinfest
economic threshold
eradication
exclusion
fallow
fumigant
fungicide
general resistance
genetic engineering
green manure
hyperparasite
hypovirulence
indexing
infection period
initial inoculum
integrated pest management (IPM)
LD_{50}
meristem
monocyclic
multiline
mycovirus
nematicide
phylloplane-competent
phytosanitary certificate
phytotoxicity
polycyclic
primary inoculum
protectant fungicide
protection

pyramiding
quarantine
rhizosphere-competent
rogue
sanitation
secondary inoculum
solarization
specific resistance
stabilizing selection
suppressive soil
systemic acquired resistance (SAR)
systemic fungicide
thermotherapy
tolerance
volunteer

Exercises

Exercise 11.1. Choose three diseases from the APS Education Center Plant Disease Profiles. At least one disease should be monocyclic and at least one should be polycyclic.

If the disease is monocyclic:

a. What is the primary inoculum?
b. What management practices are recommended? Do they reduce the amount or efficacy of primary inoculum?
c. Are any prediction or forecasting systems available?
d. Is a biological control recommended? If one is not available, what potential kinds might be useful?

If the disease is polycyclic:

a. What is the primary inoculum?
b. What is the secondary inoculum?

c. What management practices are recommended? Which are aimed at the primary inoculum and which help reduce the rate of the epidemic?
d. Is a biological control recommended? If one is not available, what potential kinds might be useful?
e. Is fungicide control recommended? What kinds of fungicides are effective? Is there a prediction system based on infection periods?

Internet Resources

APS Resources

Alternatives to methyl-bromide: A Florida perspective
Antibiotic use for plant disease management in the U.S.
Biological control of plant pathogens: Research, application and commercialization in the USA
Burkholderia cepacia: Friend or foe?
Cultivar mixtures
Population genetics of plant pathogens
Q_oI/Strobilurin fungicides: Benefits and risks
A short history of fungicides
Small grain cereal seed treatment
The future world food situation and the role of plant diseases
Transgenic virus resistant papaya: New hope for controlling *Papaya ringspot virus* in Hawaii
What are the nonindigenous plant pathogens that threaten U.S. crops and forests?

Recommended Websites

Biorationals: Ecological Pest Management Database
Fungicide Resistance Action Committee (FRAC)

Fungicide Resistance Action Committee (FRAC) Code List
NAPPO Phytosanitary Alert System
ipmPIPE
Pew Institute Factsheet on Genetically Modified Crops in the U.S.
The Future Role of Pesticides in US Agriculture, National Academy of Science Press, 2000
The Value of Fungicides in U.S. Farming Systems
Traditional Practices for Plant Disease Management in Traditional Farming Systems
US-EPA Biopesticides
US-EPA Pesticides and Food
World Health Organization—Food Safety

Internet Research

Internet Research Exercise 11.1. Choose the 10 most important diseases of a plant that you wish to grow or manage. Find information on the management of each disease. Answer these questions for each disease:

a. Is the disease monocyclic or polycyclic?
b. What management practices are recommended?
c. Which management practices focus on reducing the amount or efficacy of primary inoculum?
d. If the disease is polycyclic, what rate-reducing practices are recommended?
e. Is there a forecasting or prediction system?
f. What kinds of fungicides should you plan to use, and what is your fungicide resistance management plan?
g. Are any resistant cultivars recommended?
h. Are any biological controls available?

How could you integrate the management required for these 10 diseases into a crop/plant management plan? Do you see any management conflicts among the diseases? Are any of the management practices effective for more than one disease?

Internet Research Exercise 11.2. Go to one of these biopesticide websites: Biopesticide Active Ingredient or Biorationals: Ecological Pest Management Database. Choose a biocontrol product for a plant disease. Using the information on this website and others, try to find information about

a. the biological control organism and its mode(s) of action in this product
b. the disease(s) for which it is used
c. published research reports on its efficacy
d. any safety concerns

Internet Research Exercise 11.3. In April 2000, *Reason* magazine interviewed Nobel Laureate Norman Borlaug about the future of agriculture and our ability to feed the world in the future. Using your knowledge of plant pathology, read the article and critique this interview with Dr. Borlaug.

APPENDIX 1

Disease Classics

Disease name	Type of pathogen	Name of pathogen(s)
Anthracnose of turfgrass	fungus, ascomycete	*Colletotrichum graminicola*
Apple scab	fungus, ascomycete	*Venturia inaequalis*
Armillaria root disease	fungus, basidiomycete	*Armillaria mellea*
Bacterial spot of pepper and tomato	bacterium	*Xanthomonas axonopodis* pv. *vesicatoria*, syn. *X. campestris* pv. *vesicatoria*
Barley yellow dwarf	virus	*Barley yellow dwarf virus* (BYDV); *Cereal yellow dwarf virus* (CYDV)
Black Sigatoka of banana and plantain	fungus, ascomycete	*Mycosphaerella fijiensis*
Brown rot of stone fruits	fungus, ascomycete	*Monilinia fructicola; M. laxa*
Cedar-apple rust	fungus, basidiomycete	*Gymnosporangium juniperi-virginianae*

Chestnut blight	fungus, ascomycete	*Cryphonectria parasitica*
Citrus canker	bacterium	*Xanthomonas axonopodis* pv. *citri; X. axonopodis* pv. *aurantifolii*
Clubroot of brassicas (crucifers)	plasmodiophoromycete	*Plasmodiophora brassicae*
Coffee rust	fungus, basidiomycete	*Hemileia vastatrix*
Common smut of corn	fungus, basidiomycete	*Ustilago maydis*
Crown gall	bacterium	*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*
Dodder	parasitic plant	*Cuscuta* spp.
Downy mildew of grape	oomycete	*Plasmopara viticola*
Dutch elm disease	fungus, ascomycete	*Ophiostoma novo-ulmi; O. ulmi*
Dwarf mistletoe	parasitic plant	*Arceuthobium* spp.
Ergot of cereals and grasses	fungus, ascomycete	*Claviceps purpurea*
Fire blight	bacterium	*Erwinia amylovora*
Gray mold	fungus, ascomycete	*Botrytis cinerea*
Late blight	oomycete	*Phytophthora infestans*
Lesion nematode	nematode	*Pratylenchus* spp.

disease		
Powdery mildew of cereals and grasses	fungus, ascomycete	*Blumeria graminis*, syn. *Erysiphe graminis*
Root-knot nematode disease	nematode	*Meloidogyne* spp.
Soft rot	bacterium	*Pectobacterium carotovorum*, syn. *Erwinia carotovora*
Soybean cyst nematode disease	nematode	*Heterodera glycines*
Southern blight	fungus, basidiomycete	*Sclerotium rolfsii*
Stem rust of wheat and barley	fungus, basidiomycete	*Puccinia graminis* f. sp. *tritici*
Stewart's wilt of corn	bacterium	*Erwinia stewartii*, syn. *Pantoea stewartii*
Stinking smut (covered bunt) of wheat	fungus, basidiomycete	*Tilletia tritici; T. laevis*
Take-all root rot	fungus, basidiomycete	*Gaeumannomyces graminis*
Tobacco mosaic	virus	*Tobacco mosaic virus* (TMV)
Verticillium wilt	fungus, ascomycete	*Verticillium dahliae*
White pine blister rust	fungus, basidiomycete	*Cronartium ribicola*

Witchweed	parasitic plant	*Striga* spp.

APPENDIX 2

Example Diseases

The following are lists of important diseases of specific plants, representing several pathogen groups. See the *APS Common Names for Plant Diseases* for more extensive lists.

When doing Internet research, you are more likely to find detailed scientific and technical information about the disease if you search using the pathogen name as well as the disease name. In the list below, B = bacterium, F = fungus, N = nematode, O = oomycete, P = parasitic plant, V = virus, Vd = viroid.

Apple (*Malus* × *domestica*)
Anthracnose and perennial canker (*Pezicula malicorticis; Neofabraea perennans*) (F)
Apple chlorotic leaf spot (*Apple chlorotic leaf spot virus*) (ACLSV) (V)
Apple scab (*Venturia inaequalis*) (F)
Apple stem pitting (*Apple stem pitting virus*) (ASPV) (V)
Black rot (*Botryosphaeria obtusa*) (F)
Cedar-apple rust (*Gymnosporangium juniperi-virginianae*) (F)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Fire blight (*Erwinia amylovora*) (B)
Lesion nematode (*Pratylenchus* spp.) (N) (see apple replant disease)
Phytophthora crown, collar, and root rots (*Phytophthora* spp.) (O)
Powdery mildew (*Podosphaera leucotricha*) (F)

Avocado (*Persea americana*)
Anthracnose (*Colletotrichum gloeosporioides*) (F)

Bacterial canker (*Pseudomonas syringae, Xanthomonas campestris*) (B)
Blast and bacterial fruit spot (*Pseudomonas syringae*) (B)
Fruit rot (includes stem end rot and fruit spots) (*Botryodiplodia* sp., *Botryosphaeria obtusa*) (F)
Lesion nematode (*Pratylenchus vulnus*) (N)
Phytophthora crown rot, trunk canker, root rot (*Phytophthora cinnamomi, P. citricola*) (O)
Sunblotch (*Avocado sunblotch viroid*) (ASBVd) (Vd)
Verticillium wilt (*Verticillium dahliae*) (F)

Banana and plantain (*Musa* spp.)
Anthracnose and crown rot (*Colletotrichum musae*) (F)
Bacterial wilt (*Ralstonia solanacearum* Race 1, syn. *Pseudomonas solanacearum* Race 1) (B)
Black leaf streak (black Sigatoka) (*Mycosphaerella fijiensis*) (F)
Blackhead toppling syndrome (burrowing nematode) (*Radopholus similis*) (N)
Bunchy top (*Banana bunchy top virus*) (BBTV) (V)
Emusae leaf spot (*Mycosphaerella eumusae*) (F)
Fusarium wilt (Panama disease) (*Fusarium oxysporum* f. sp. *cubense*) (F)
Moko (*Ralstonia solanacearum* Race 2, syn. *Pseudomonas solanacearum* Race 2) (B)
Streak (*Banana streak virus*) (BSV) (V)
(Yellow) Sigatoka (*Mycosphaerella musicola*) (F)

Bean (*Phaseolus vulgaris*)
Angular leaf spot (*Phaeoisariopsis griseola*) (F)
Anthracnose (*Colletotrichum lindemuthianum*) (F)
Bean common mosaic (*Bean common mosaic virus*) (BCMV) (V)
Bean yellow mosaic (*Bean yellow mosaic virus*) (BYMV) (V)
Common bean blight (*Xanthomonas axonopodis* pv. *phaseoli*) (B)
Damping-off, stem rot, and root rot (*Rhizoctonia solani*) (F)
Gray mold (*Botrytis cinerea*) (F)

Halo blight (*Pseudomonas syringae* pv. *phaseolicola*) (B)
Lesion nematode (*Pratylenchus* spp.) (N)
Root-knot nematode (*Meloidogyne* spp.) (N)
Rust (*Uromyces appendiculatus*) (F)
Southern blight (*Sclerotium rolfsii*) (F)
Web blight (*Thanatephorus cucumeris*) (F)
White mold (*Sclerotinia sclerotiorum*) (F)

Brassicas/Crucifers (*Brassica* spp.; *Raphanus* spp.)
Alternaria diseases: black spot (leaf, stem, or pod spots) (*Alternaria* spp., e.g., *A. brassicae*) (F)
Black leg and Phoma root rot (*Leptosphaeria maculans*; anamorph: *Phoma lingam*) (F)
Black rot (*Xanthomonas campestris* pv. *campestris*) (B)
Cauliflower mosaic (*Cauliflower mosaic virus*) (CaMV) (V)
Clubroot (*Plasmodiophora brassicae*) (plasmodiophoromycete)
Downy mildew, staghead (*Peronospora parasitica*, syn. *Hyaloperonospora parasitica*) (O)
Mosaic (*Cauliflower mosaic virus; Turnip mosaic virus*) (CaMV; TuMV) (V)
Sugar beet cyst nematode (*Heterodera schachtii*) (N)
White mold (*Sclerotinia sclerotiorum*) (F)
White rust (*Albugo candida*) (O)
Yellows (*Fusarium oxysporum* f. sp. *conglutinans*) (F)

Cacao (*Theobroma cacao*)
Black pod and stem canker (*Phytophthora* spp.) (O)
Cacao swollen shoot (*Cacao swollen shoot virus* (CSSV) (V)
Ceratocystis wilt (*Ceratocystis fimbriata*) (F)
Frosty pod rot (*Crinipellis roreri*) (F)
Pink disease (*Erythricium salmonicolor*) (F)
Root-knot nematode (*Meloidogyne incognita*) (N)
Vascular streak dieback (*Oncobasidium theobromae*) (F)
Witches' broom (*Crinipellis perniciosa*) (F)

Chrysanthemum
Bacterial blight (*Erwinia chrysanthemi*) (B)
Bacterial leaf spot (*Pseudomonas cichorii*) (B)
Botrytis blight (*Botrytis cinerea*) (F)
Brown rust (*Puccinia chrysanthemi*) (F)
Chrysanthemum stunt (*Chrysanthemum stunt viroid*) (CSVd) (Vd)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium rhizobacter*) (B)
Foliar nematode (*Aphelenchoides* spp.) (N)
Fusarium wilt (*Fusarium oxysporum* f. sp. *chrysanthemi*) (F)
Impatiens necrotic spot (*Impatiens necrotic spot virus*) (INSV) (V)
Mosaic (*Cucumber mosaic virus*) (CMV) (V)
Powdery mildew (*Erysiphe cichoracearum*) (F)
Pythium root rot (*Pythium* spp.) (O)
Rhizoctonia crown rot (*Rhizoctonia solani*) (F)
Tomato spotted wilt (*Tomato spotted wilt virus*) (TSWV) (V)
Verticillium wilt (*Verticillium albo-atrum; V. dahliae*) (F)
White rust (*Puccinia horiana*) (F)

Citrus
Canker (*Xanthomonas axonopodis* pv. *citri; X. axonopodis* pv. *aurantifolii*) (B)
Foot rot, gummosis, root rot (*Phytophthora* spp.) (O)
Fruit decays: blue mold, green mold, whisker mold (*Penicillium* spp.) (F)
Greasy spot (*Mycosphaerella citri*) (F)
Huanglongbing (greening) (*Candidatus* Liberibacter asiaticus; *Candidatus* L. africanus) (B)
Scab (*Elsinoe fawcettii*) (F)
Slow decline (*Tylenchulus semipenetrans*) (N)
Stubborn (*Spiroplasma citri*) (B)
Tristeza (*Citrus tristeza virus*) (CTV) (V)

Coconut palm (*Cocos nucifera*)

Bud rot (*Fusarium moniliforme*) (F)
Bud rot and premature nut fall (*Phytophthora palmivora*) (O)
Bud and root rot (*Fusarium solani*) (F)
Bud rot, leaf spot, heart rot, stem bleeding and root decay (*Thielaviopsis paradoxa*) (F)
Butt rot, basal stem rot (*Ganoderma boninense*) (F)
Cadang-cadang (Coconut cadang-cadang viroid) (CCCVd) (Vd)
Leaf spot (*Bipolaris incurvata*) (F)
Lethal yellowing (phytoplasma) (B)
Petiole and stem rot (*Fusarium oxysporum*) (F)
Red ring (*Bursaphelenchus cocophilus*) (N)
Root rot (*Rhizoctonia solani*) (F)

Coffee (*Coffea arabica, C. canephora*)
Anthracnose (*Colletotrichum gloeosporioides*) (F)
Bacterial blight (*Pseudomonas syringae* pv. *garcae*) (B)
Coffee berry disease (*Colletotrichum kahawae* (*coffeanum*)) (F)
Root knot (*Meloidogyne* spp.) (N)
Rust (orange or leaf rust) (*Hemileia vastatrix*) (F)
Wilt (*Ceratocystis fimbriata*) (F)

Conifers
Annosum root rot (*Heterobasidion annosum*, syn. *Fomes annosum*) (F)
Armillaria root disease (*Armillaria* spp.) (F)
Black-stain root disease (*Leptographium wageneri*) (F)
Dwarf mistletoe (*Arceuthobium* spp.) (P)
Eastern gall rust (*Cronartium quercuum* f. sp. *banksianae*) or Western gall rust (*Endocronartium harknessii*) (F)
Fusiform rust (*Cronartium quercuum* f. sp. *fusiforme*) (F)
Phytophthora root rot (littleleaf disease) (*Phytophthora cinnamomi*) (O)
Pine wilt (*Bursaphelenchus xylophilus*) (N)
Pitch canker (*Fusarium subglutinans*) (F)
Red ring rot (heart rot) (*Phellinus pini*, syn. *Fomes pini*) (F)

Rhabdocline needle cast (*Rhabdocline pseudotsugae*, syn. *R. weireii*) (F)
Sphaeropsis shoot blight and canker (*Sphaeropsis sapinea*) (F)
White pine blister rust (*Cronartium ribicola*) (F)

Corn (*Zea mays*)
Common rust (*Puccinia sorghi*) (F)
Common smut (*Ustilago maydis*) (F)
Corn stunt (*Spiroplasma kunkelii*) (B)
Downy mildew (*Peronosclerospora* spp.; *Sclerospora* spp.) (O)
Gibberella stalk rot (*Gibberella zeae*; anamorph: *Fusarium graminearum*) (F)
Gray leaf spot (*Cercospora zeae-maydis*) (F)
Lesion nematode (*Pratylenchus* spp.) (N)
Maize dwarf mosaic (*Maize dwarf mosaic virus*) (MDMV) (V)
Southern corn leaf blight (*Cochliobolus heterostrophus*; anamorph: *Bipolaris maydis*) (F)
Stewart's bacterial wilt (*Erwinia stewartii*, syn. *Pantoea stewartii*) (B)
Witchweed (*Striga* spp.) (P)

Cucurbits (*Citrullus* spp., *Cucumis* spp., *Cucurbita* spp., and others)
Angular leaf spot (*Pseudomonas syringae* pv. *lachrymans*) (B)
Anthracnose (*Colletotrichum orbiculare*) (F)
Bacterial wilt (*Erwinia tracheiphila*) (B)
Cucumber mosaic (*Cucumber mosaic virus*) (CMV) (V)
Downy mildew (*Pseudoperonospora cubensis*) (O)
Fusarium wilt (*Fusarium oxysporum* f. sp. *cucumerinum*; *F. oxysporum* f. sp. *melonis*, etc.) (F)
Gummy stem blight and black rot (*Didymella bryoniae*) (F)
Papaya ringspot (*Papaya ringspot virus*) (PRSV) (V)
Phytophthora blight (*Phytophthora* spp.) (O)
Powdery mildew (*Podosphaera xanthii*, syn. *Sphaerotheca fuliginea; Erysiphe cichoracearum*) (F)

Root-knot nematode (*Meloidogyne* spp.) (N)
Watermelon mosaic (*Watermelon mosaic virus*) (WMV) (V)
Zucchini yellow mosaic (*Zucchini yellow mosaic virus*) (ZYMV) (V)

Geranium (*Pelargonium*)
Bacterial blight (*Xanthomonas campestris* pv. *pelargonii*) (B)
Bacterial leaf spot (*Acidovorax* spp.; *Pseudomonas* spp.) (B)
Bacterial wilt (*Ralstonia solanacearum* Race 1 and Race 3, syn. *Pseudomonas solanacearum* Race 1 and Race 3) (B)
Black root rot (*Thielaviopsis basicola*) (F)
Botrytis blight (*Botrytis cinerea*) (F)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Pelargonium flower break (*Pelargonium flower break virus*) (PFBV) (V)
Pythium root rot (*Pythium* spp.) (O)
Rhizoctonia root and crown rot (*Rhizoctonia solani*) (F)
Rust (*Puccinia pelargonii-zonalis*) (F)
Verticillium wilt (*Verticillium albo-atrum; V. dahliae*) (F)

Grape (*Vitis* spp.)
Black rot (*Guignardia bidwellii*) (F)
Botrytis bunch rot and blight (*Botrytis cinerea*) (F)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Dagger nematode (*Xiphinema* spp.) (N) (vector of *Grapevine fanleaf virus*, GFLV)
Downy mildew (*Plasmopara viticola*) (O)
Eutypa dieback (*Eutypa lata*) (F)
Fanleaf degeneration (*Grapevine fanleaf virus*) (GFLV) (V)
Pierce’s disease (*Xylella fastidiosa*) (B)
Powdery mildew (*Uncinula necator*) (F)

Papaya (*Carica papaya*)
Anthracnose (*Colletotrichum gloeosporioides*) (F)

Bacterial crown rot (*Erwinia cariceae*) (B)
Bacterial wilt (*Ralstonia solanacearum*, syn. *Pseudomonas solanacearum*) (B)
Black spot (*Asperisporium caricae, Cercospora papayae*) (F)
Brown spot (*Corynespora cassiicola*) (F)
Papaya ringspot (*Papaya ringspot virus*) (PRSV) (V)
Powdery mildew (*Erysiphe cichoracearum*) (F)
Reniform nematode (*Rotylenchulus reniformis, R. parvus*) (N)
Root and fruit rot (*Phytophthora palmivora*) (O)
Root knot (*Meloidogyne* spp.) (N)
Spotted wilt (*Tomato spotted wilt virus*) (TSWV) (V)

Peanut (*Arachis hypogaea*)
Bacterial wilt (*Ralstonia solanacearum* Race 1, syn. *Pseudomonas solanacearum* Race 1) (B)
Bud necrosis (*Groundnut bud necrosis virus*) (GBNV) (V)
Dodder (*Cuscuta campestris*) (P)
Leaf spot, early (*Cercospora arachidicola*; teleomorph: *Mycosphaerella arachidis*) (F)
Leaf spot, late (*Phaeoisariopsis personata*, syn. *Cercosporidium personatum*; teleomorph: *Mycosphaerella berkeleyi*) (F)
Lesion nematode (*Pratylenchus brachyurus; P. coffeae*) (N)
Peanut mottle (*Peanut mottle virus*) (PeMoV) (V)
Pythium pod rot (*Pythium myriotylum*) (O)
Rhizoctonia foliar blight, limb, peg, and root rot (*Rhizoctonia solani*) (F)
Root-knot nematode (*Meloidogyne javanica; M. hapla; M. arenaria*) (N)
Rust (*Puccinia arachidis*) (F)
Sclerotinia blight (*Sclerotinia minor; S. sclerotiorum*) (F)
Stem rot (southern blight) (*Sclerotium rolfsii*; teleomorph: *Athelia rolfsii*) (F)
Tomato spotted wilt (*Tomato spotted wilt virus*) (TSWV) (V)

Verticillium wilt (*Verticillium albo-atrum*; *V. dahliae*) (F)
Witches' broom (phytoplasma) (B)
Yellow mold (aflatoxin) (*Aspergillus flavus*; *A. parasiticus*) (F)

Potato (*Solanum tuberosum*)
Aster yellows (aster yellows group of phytoplasmas) (B)
Bacterial ring rot (*Clavibacter michiganensis* subsp. *sepedonicus*) (B)
Bacterial wilt, syn. brown rot (*Ralstonia solanacearum* Race 1 and Race 3, syn. *Pseudomonas solanacearum* Race 1 and Race 3) (B)
Black dot (*Colletotrichum coccodes*) (F)
Blackleg and bacterial soft rot (*Pectobacterium carotovorum* subsp. *atrosepticum*, syn. *Erwinia carotovora* subsp. *atroseptica*) (B)
Common rust (*Puccinia pittieriana*) (F)
Common scab (*Streptomyces scabiei*, syn. *S. scabies; S. acidiscabie; S. turgidiscabies*) (B)
Early blight (*Alternaria solani*) (F)
Fusarium wilt (*Fusarium oxysporum*, several formae speciales) (F)
Gray mold (*Botrytis cinerea*) (F)
Late blight (*Phytophthora infestans*) (O)
Lesion nematode (*Pratylenchus* spp.) (N)
Potato cyst, syn. golden nematode (*Globodera rostochiensis; G. pallida*) (N)
Potato leafroll virus (*Potato leafroll virus*) (PLRV) (V)
Potato spindle tuber (*Potato spindle tuber viroid*) (PSTVd) (Vd)
Potato virus X (*Potato virus X*) (PVX) (V)
Potato virus Y (*Potato virus Y*) (PVY) (V)
Powdery scab (*Spongospora subterranea*) (plasmodiophoromycete)
Rhizoctonia canker and black scurf (*Rhizoctonia solani*) (F)
Root-knot nematode (*Meloidogyne* spp.) (N)
Silver scurf (*Helminthosporium solani*) (F)
Southern blight or stem rot (*Sclerotium rolfsii*; teleomorph: *Athelia rolfsii*) (F)
Verticillium wilt (*Verticillium albo-atrum; V. dahliae*) (F)

Wart (*Synchytrium endobioticum*) (F)
White mold (*Sclerotinia sclerotiorum*) (F)

Rice (*Oryza sativa*)
Bacterial blight (*Xanthomonas oryzae* pv. *oryzae*, syn. *X. campestris* pv. *oryzae*) (B)
Bakanae, syn. foolish seedling (*Gibberella fujikuroi*; anamorph: *Fusarium moniliforme*) (F)
Blast (leaf, neck [rotten neck], nodal, and collar) (*Pyricularia grisea*, syn. *P. oryzae*; teleomorph: *Magnaporthe grisea*) (F)
Brown spot (*Cochliobolus miyabeanus*; anamorph: *Bipolaris oryzae*) (F)
Kernel smut (*Tilletia barclayana*, syn. *Neovossia horrida*) (F)
Rice-root nematode (*Hirschmanniella* spp.) (N)
Root-knot nematode (*Meloidogyne* spp.) (N)
Sheath blight (*Thanatephorus cucumeris*; anamorph: *Rhizoctonia solani*) (F)
Stem rot (*Magnaporthe salvinii*; synanamorphs: *Sclerotium oryzae, Nakataea sigmoidae*) (F)
Tungro (*Rice tungro spherical virus*) (RTSV) (V)
White tip (*Aphelenchoides besseyi*) (N)
Yellow dwarf (phytoplasma) (B)

Rose (*Rosa* spp.)
Black spot (*Diplocarpon rosae*) (F)
Botrytis blight (*Botrytis cinerea*) (F)
Brand canker (*Coniothyrium wernsdorffiae*) (F)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Cylindrocladium root rot (*Cylindrocladium scoparium*) (F)
Downy mildew (*Peronospora sparsa*) (O)
Powdery mildew (*Sphaerotheca pannosa* var. *rosae*) (F)
Root-knot nematode (*Meloidogyne* spp.) (N)
Rust (*Phragmidium* spp.) (F)

Verticillium wilt (*Verticillium albo-atrum; V. dahliae*) (F)

Soybean (*Glycine max*)

Bacterial blight (*Pseudomonas savastanoi* pv. *glycinea*) (B)
Bean pod mottle (*Bean pod mottle virus*) (BPMV) (V)
Brown stem rot (*Phialophora gregata*) (F)
Bud blight (*Tobacco ringspot virus*) (TRSV) (V)
Charcoal rot (*Macrophomina phaseolina*) (F)
Phytophthora rot (*Phytophthora sojae*) (O)
Rhizoctonia damping-off and root rot (*Rhizoctonia solani*) (F)
Rust (*Phakopsora meibomiae; P. pachyrrhizi*) (F)
Sclerotinia stem rot (*Sclerotinia sclerotiorum*) (F)
Soybean cyst nematode (*Heterodera glycines*) (N)
Soybean mosaic (*Soybean mosaic virus*) (SMV) (V)
Stem canker (*Diaporthe phaseolorum* var. *meridionalis* and var. *caulivora*) (F)
Sudden death syndrome (*Fusarium solani* f. sp. *glycines*) (F)

Stone fruits (apricot, cherry, nectarine, peach, plum) (*Prunus* spp.)

Armillaria root rot (*Armillaria mellea*) (F)
Bacterial canker (*Pseudomonas syringae*) (B)
Black knot (*Apiosporina morbosa*, syn. *Dibotryon morbosum*) (F)
Brown rot (*Monilinia fructicola; M. laxa*) (F)
Cherry leaf spot (*Blumeriella jaapii*, syn. *Coccomyces hiemalis*) (F)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Leaf curl (*Taphrina deformans*) (F)
Leucostoma canker (*Leucostoma cincta*, syn. *Valsa cincta*) (F)
Peach yellows and X disease (phytoplasma) (B)
Phytophthora root and crown rots (*Phytophthora* spp.) (O)
Plum pox (*Plum pox virus*) (PPV) (V)
Prunus necrotic ringspot (*Prunus necrotic ringspot virus*) (PNRSV) (V)

Silver leaf disease (*Chondrosterium purpureum*, syn. *Stereum purpureum*) (F)

Strawberry (*Fragaria* × *ananassa*)

Anthracnose crown rot, fruit rot (*Colletotrichum fragariae* and other *Colletotrichum* spp.) (F)
Botrytis fruit rot (gray mold) and blossom blight (*Botrytis cinerea*) (F)
Lesion nematode (*Pratylenchus* spp.) (N)
Powdery mildew (*Sphaerotheca macularis* f. sp. *fragariae*) (F)
Red stele root rot (*Phytophthora fragariae* var. *fragariae*) (O)
Strawberry aster yellows (phytoplasma) (B)
Strawberry mottle (*Strawberry mottle virus*) (SMoV) (V)
Tomato ringspot (*Tomato ringspot virus*) (ToRSV) (V)
Verticillium wilt (*Verticillium albo-atrum*; *V. dahliae*) (F)

Sugarcane

Downy mildew (*Peronosclerospora sacchari*) (O)
Eye spot (*Bipolaris sacchari*) (F)
Leaf scald (*Xanthomonas albilineans*) (B)
Lesion nematode (*Pratylenchus zeae*) (N)
Mosaic (*Sugarcane mosaic virus*) (SCMV) (V)
Pineapple disease (*Ceratocystis paradoxa*) (F)
Pokkah boeng (*Gibberella fujikuroi*; anamorph *Fusarium moniliforme*) (F)
Ratoon stunting disease (*Clavibacter xyli* subsp. *xyli*) (B)
Red rot (*Glomerella tucumanensis*) (F)
Rust, common (*Puccinia melanocephala*) (F)
Smut, culmicolous (*Ustilago scitaminea*) (F)
Sugarcane yellow leaf virus (ScYLV) (V)

Tomato (*Lycopersicon esculentum*)

Anthracnose (*Colletotrichum coccodes*; *C. dematium*; *C. gloeosporioides*; teleomorph: *Glomerella cingulata*) (F)
Aster yellows (aster yellows phytoplasma) (B)

Bacterial canker (*Clavibacter michiganensis* subsp. *michiganensis*) (B)
Bacterial speck (*Pseudomonas syringae* pv. *tomato*) (B)
Bacterial spot (*Xanthomonas campestris* pv. *vesicatoria*) (B)
Bacterial wilt (*Ralstonia solanacearum* Race 1 and Race 3, syn. *Pseudomonas solanacearum* Race 1 and Race 3) (B)
Buckeye fruit and root rot (*Phytophthora capsici* and other *Phytophthora* spp.) (O)
Common mosaic of tomato (internal browning of fruit) (*Tobacco mosaic virus*) (TMV) (V)
Cucumber mosaic (*Cucumber mosaic virus*) (CMV) (V)
Early blight (*Alternaria solani*; *A. tomatophilia*) (F)
Fusarium wilt (*Fusarium oxysporum* f. sp. *lycopersici*) (F)
Late blight (*Phytophthora infestans*) (O)
Leaf mold (*Fulvia fulva*) (F)
Powdery mildew (*Oidiopsis sicula*; teleomorph: *Leveillula taurica*; also *Oidium neolycopersici*) (F)
Root-knot nematode (*Meloidogyne* spp.) (N)
Southern blight (*Sclerotium rolfsii*; teleomorph: *Athelia rolfsii*) (F)
Tomato spotted wilt (*Tomato spotted wilt virus*) (TSWV) (V)
Tomato yellow leaf curl (*Tomato yellow leaf curl virus*) (TYLCV) (V)
Verticillium wilt (*Verticillium albo-atrum*; *V. dahliae*) (F)
White mold (*Sclerotinia sclerotiorum*) (F)

Turfgrasses
Anthracnose (*Colletotrichum graminicola*) (F)
Brown patch (*Rhizoctonia solani*) (F)
Dollar spot (*Sclerotinia homoeocarpa*) (F)
Fairy ring (various basidiomycetes) (F)
Gray leaf spot (*Magnaporthe grisea*; anamorph: *Pyricularia grisea*) (F)
Leaf spot and crown and root rot (*Bipolaris* spp.; *Drechslera* spp.) (F)
Necrotic ring spot of *Poa* spp. (*Ophiosphaerella korrae*) (F)
Nematodes (many, especially sting nematode, *Belonolaimus* spp.) (N)

Pythium blight (*Pythium aphanidermatum* and other *Pythium* spp.) (O)

Red thread (*Laetisaria fuciformis*) (F)

Rust (*Puccinia* spp.) (F)

Snow mold (*Typhula* spp.; *Microdochium nivale*) (F)

Spring dead spot of bermudagrass (*Ophiosphaerella korrae* and others) (F)

Summer patch of *Poa* spp. (*Magnaporthe poae*) (F)

Take-all patch of bentgrass (*Gaeumannomyces graminis* var. *avenae*) (F)

Wheat (*Triticum* spp.)

Aster yellows (aster yellows phytoplasma) (B)

Bacterial leaf blight (*Pseudomonas syringae* subsp. *syringae*) (B)

Barley yellow dwarf (*Barley yellow dwarf virus*) (BYDV) (V)

Cereal cyst nematode (*Heterodera avenae*) (N)

Common bunt, syn. stinking smut (*Tilletia tritici*) (F)

Downy mildew, syn. crazy top (*Sclerophthora macrospora*) (O)

Loose smut (*Ustilago tritici*) (F)

Powdery mildew (*Erysiphe graminis* sp. *tritici*, syn. *Blumeria graminis*) (F)

Scab, syn. head blight (*Fusarium* spp.; teleomorph: *Gibberella zeae*) (F)

Seed gall (wheat gall) nematode (*Anguina tritici*) (N)

Septoria blotch (*Mycosphaerella graminicola*; anamorph: *Septoria tritici*) (F)

Soil-borne wheat mosaic (*Soil-borne wheat mosaic virus*) (SBWMV) (V)

Stem rust, syn. black rust (*Puccinia graminis* f. sp. *tritici*) (F)

Storage mold (*Aspergillus* spp.; *Penicillium* spp.) and others (F)

Stripe rust, syn. yellow rust (*Puccinia striiformis*) (F)

Take-all (*Gaeumannomyces graminis* var. *tritici*) (F)

Wheat streak mosaic (*Wheat streak mosaic virus*) (WSMV) (V)

Witchweed (*Striga* spp.) (P)

Woody ornamentals

Anthracnose of sycamore, maple, and oak (*Apiognomonia* spp.) (F)
Armillaria root disease (*Armillaria* spp.) (F)
Bacterial leaf scorch of shade trees (sycamore, elm, oak, etc.) (*Xylella fastidiosa*) (B)
Black spot of rose (*Diplocarpon rosae*) (F)
Cedar-apple rust of junipers and rosaceous hosts (*Gymnosporangium juniperi-virginianae*)
Crown gall (*Agrobacterium tumefaciens*, syn. *Rhizobium radiobacter*) (B)
Diplodia tip blight of conifers (*Sphaeropsis sapinea*) (F)
Dutch elm disease (*Ophiostoma novo-ulmi; O. ulmi*) (F)
Fire blight of rosaceous ornamentals (*Erwinia amylovora*) (B)
Lethal yellowing of palm (phytoplasma) (B)
Nectria canker of hardwoods (*Nectria galligena*) (F)
Phytophthora root rot and Ramorum blight (sudden oak death) (*Phytophthora* spp.) (O)
Pine wilt (*Bursaphelenchus xylophilus*) (N)
Powdery mildew (species of *Erysiphe, Microsphaera, Phyllactinia, Podosphaera, Sphaerotheca, Uncinula*) (F)
Verticillium wilt (*Verticillium albo-atrum*; *V. dahliae*) (F)

APPENDIX 3

Rapid Assays for Pathogens

Although some diseases can be diagnosed by observing pathogens in infected plant tissues (e.g., fungi, some nematodes), most require some kind of laboratory culturing. This is most often done for common bacterial and fungal diseases, but it may require days to weeks to complete and significant diagnostic expertise to identify and interpret the results. In addition, many pathogens are obligate parasites (fastidious bacteria, some fungi, nematodes, viruses) that are difficult or impossible to isolate in culture. Two kinds of tests are commonly used for identification of pathogens in plant tissue: **immunoassays** and **polymerase chain reactions** (**PCR**s). Immunoassays or **serological tests** are based on the specificity of the antigen-antibody reaction in animal immune systems. PCR tests are based on the identification of certain sequences of nucleic acids (DNA or RNA). Both assays can be completed quickly and do not require culturing. However, one of the most important limitations of these rapid assays is that users must know what pathogen(s) they are trying to detect.

Immunoassays

Serological tests, or immunoassays, have been used for viral disease diagnosis for more than 50 years. These tests rely on identification of a pathogen (containing an **antigen**) through its reaction with specific antibodies. **Antibodies** are molecules produced in animals by their immune systems as a defense against foreign macromolecules, including viral proteins and, to a lesser extent, nucleic acids. Plant pathologists inject purified preparations of pathogens into laboratory animals to induce production of specific antibodies in the blood serum (**antiserum**). After several weeks, the antibodies are harvested from blood samples taken from the animals. These antibodies bind specifically to the pathogen used to create them and, to a lesser extent, to related organisms. Usually a different antiserum must be produced for each pathogen. Some older serological tests rely on direct visualization of the antigen-antibody reaction, usually in the form of a precipitate, and a successful test requires high concentrations of the pathogen to produce a visible reaction. More recently developed serological tests do not rely on the visualization of a precipitate, and therefore can detect lower concentrations of a pathogen.

Immunoassays

- based on the uniqueness of proteins on the surface of the pathogen
- require a specific antigen (pathogen)-antibody reaction
- can be used for general or specific pathogen assays
- may require a different immunoassay (different antibodies) for each pathogen
- Monoclonal antibodies allow the use of immunoassays for complex organisms with many surface proteins. They also permit tissue culture production of quantities of antibodies for affordable commercial immunoassay kits.
- ELISA tests use an enzyme “linked” to an antibody that produces a color reaction in the presence of the pathogen. This helps to detect even low concentrations of a pathogen.
- Antibodies are proteins, so immunoassay kits usually do not remain active more than 12 months. They must be refrigerated, but not frozen.

- Immunoassays can be done in the field or greenhouse, often in less than 1 hour with no special equipment.

For many decades all antisera were **polyclonal**, meaning that they contained a mixture of different antibodies that recognized different parts of the pathogen used to create them. More recently, plant pathologists have produced **monoclonal antibodies** (specific antibodies produced from a single clone, or cell) that recognize only one part of a pathogen (Figure 1). In addition to this specificity, another advantage of monoclonal antibodies is that the cells in which they are produced are hybrids of an antibody-producing cell and a cancer cell that has indefinite life. These hybrid cells (**hybridomas**) can be frozen in liquid nitrogen, revived several years later, and used to produce another batch of the same antibodies. Rather than using the blood of laboratory animals as a source of antibodies, these antibodies can be mass-produced from cell cultures. Monoclonal antibodies are now used to quickly identify viruses and more complex pathogens such as bacteria, fungi, and nematodes.

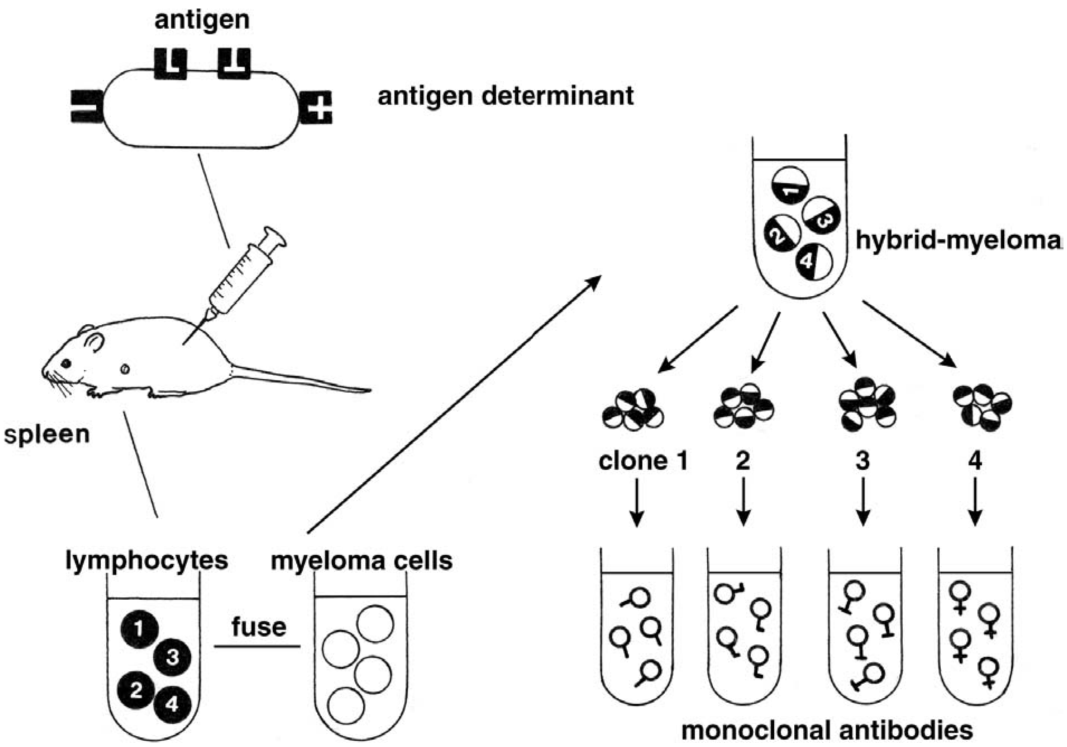

Figure 1. Steps in the process of generating monoclonal antibodies.

The most common serological test for the diagnosis of plant diseases is the **enzyme-linked immunosorbent assay** or **ELISA** (Figure 2). ELISA can use either polyclonal or monoclonal antibodies or a combination of both. There are many variants of this assay, which is usually conducted in 96-well plastic microtiter plates (Figure 3). In a common version of ELISA, called the **double-antibody sandwich (DAS)**, wells are first coated with antibodies produced against the pathogen (antigen) that the diagnostician believes may be responsible for the disease. Next, a preparation of plant tissue presumably containing the pathogen is added to each well and incubated to permit the antigen to bind to the antibodies already attached to the well. The wells are washed after each step to remove unbound reagents. A solution containing the **conjugate**, modified antibodies that can bind to sites on the antigen, is added to each well. These antibodies are conjugated, or linked, to an enzyme, often alkaline phosphatase. When the antigen has become attached to the initial coating antibodies, the conjugated antibodies will then bind to available sites on the antigen, forming a “sandwich.” After washing, an enzyme **substrate** is added that will result in color development only if the conjugate has bound to the antigen. In the case of alkaline phosphatase, a yellow color that can be measured in a spectrophotometer (an instrument that measures light intensity) develops in wells where the pathogen is present. The intensity of the yellow color is related to the pathogen concentration.

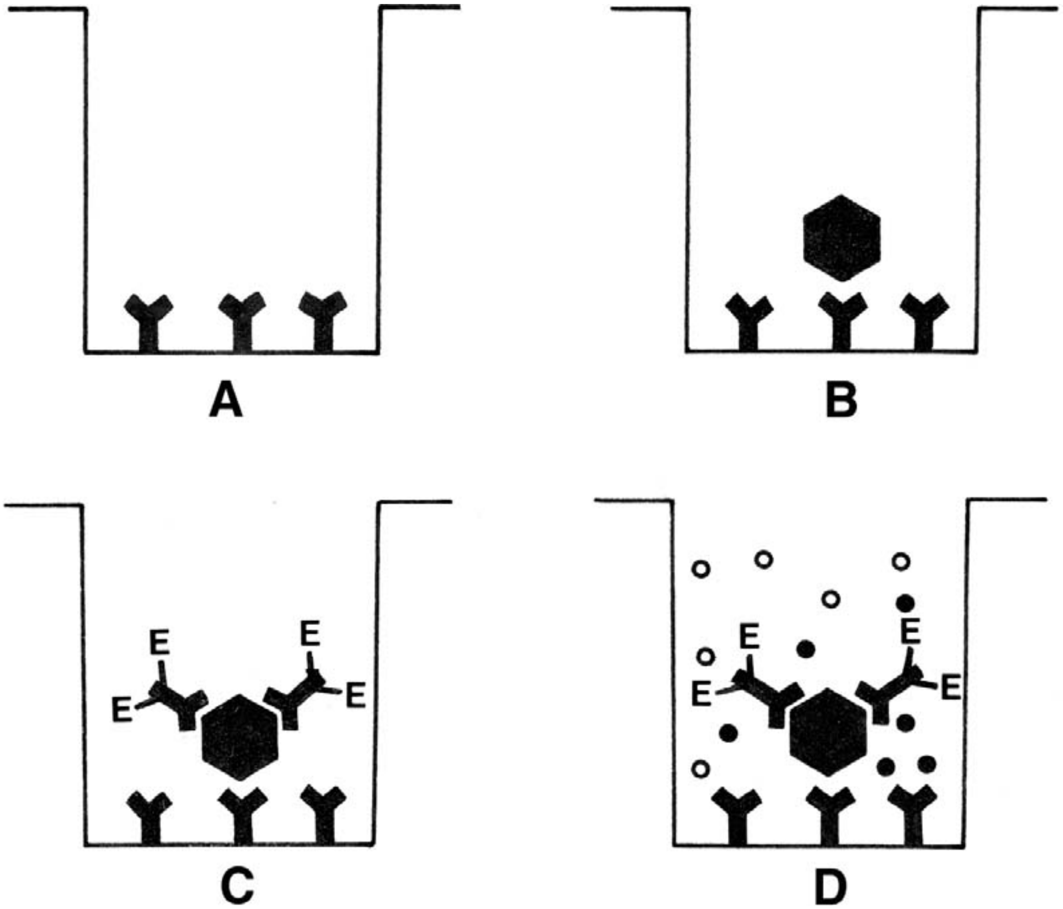

Figure 2. Procedures used for enzyme-linked immunosorbent assay (ELISA). A, A specific antibody is adsorbed to the plate, which is incubated and then washed to remove unbound materials. B, The test sample containing the pathogen (antigen) is added, and the plate is incubated and washed. C, The enzyme-labeled (E) specific antibody (conjugate) is added, and the plate is incubated and washed. D, The enzyme substrate (circles) is added and is acted upon by the enzyme to produce the colored product (dark circles).

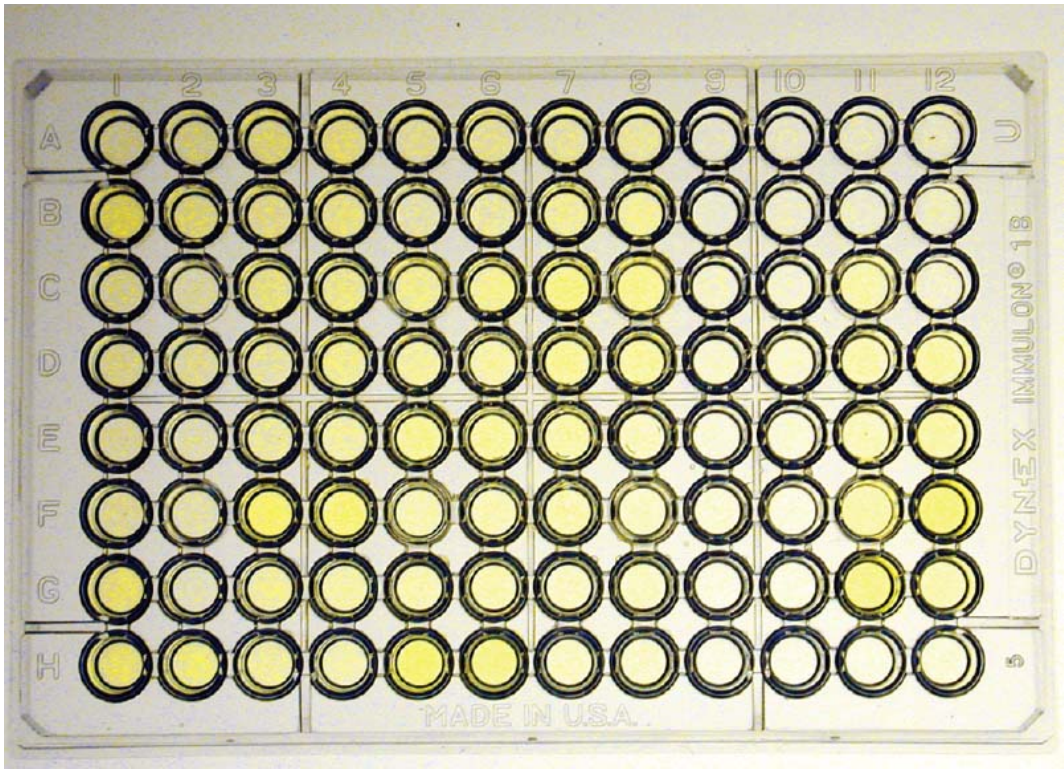

Figure 3. Multiwell plate for ELISA. Each well contains a test sample. The wells showing yellow color are positive.

Advantages of ELISA include specificity, speed, and sensitivity. Relatively low concentrations of antigen can trigger the color reaction, and this is helpful when pathogen populations in the host tissue are low or symptoms of the disease are not yet apparent. In addition, a diagnostic laboratory can test many samples in 1 to 2 days using ELISA if the necessary antisera are on hand. The assay can be automated for large-scale testing, as is required in programs that certify seed or vegetative plant parts free of certain pathogens.

Online Resource for ELISA Immunoassays (also used for rapid medical assays)

The Biology Project: Immunology, University of Arizona, ELISA animation

Other types of immunoassays also may be performed in a simpler format. For example, some assays involve grinding plant tissue in a plastic bag and then testing the resulting sap with an "immunostrip" (Figure 4). These strips have the antisera bound onto a narrow piece of plastic; the strip develops color in a horizontal band if the pathogen is present. Results are available within minutes and can be compared with a positive reaction test impregnated on the strip.

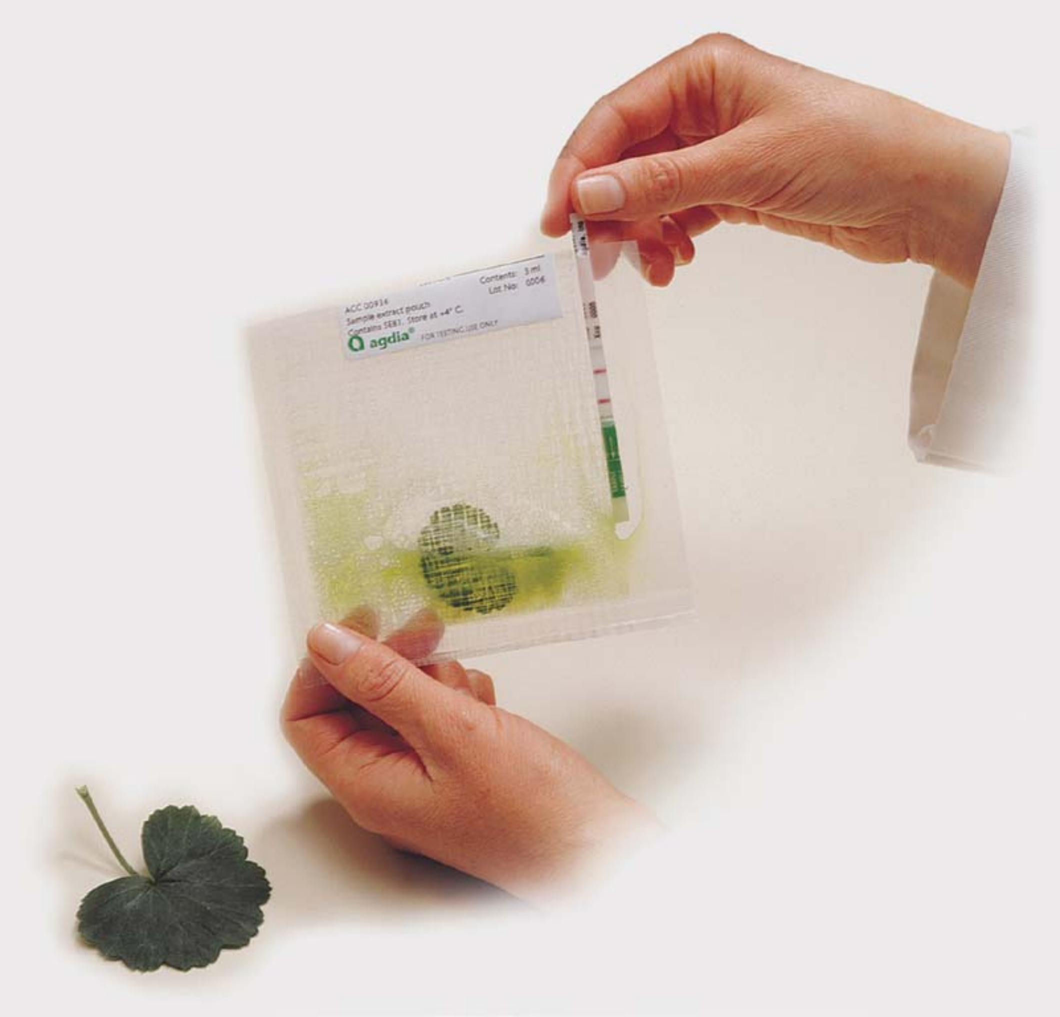

Figure 4. Immunostrips for rapid on-site pathogen identification using ELISA. Color bands form if the pathogen is present in the sample.

DNA-based tests, including PCR

Serological tests are based on the uniqueness of proteins of the pathogen. There also are diagnostic procedures based on detection of unique nucleic acids, including polyacrylamide gel electrophoresis, nucleic acid hybridization, and **polymerase chain reaction (PCR)** (Figure 5). Since the 1980s, PCR has become widely used for plant disease diagnosis because of its relative simplicity and great sensitivity. As in the use of ELISA for plant disease diagnosis, the use of PCR relies on the diagnostician's knowledge of pathogens that are likely to be the cause of the disease problem. In ELISA, this knowledge guides the selection of antibodies used in the assay; in PCR, it guides the selection of short (14–30 nucleotide) pieces of DNA, called **primers**, which can pair (**anneal**) with specific segments of the pathogen's genome. As with serological tests, different primers must be found for each pathogen, or even for strains or biotypes of a specific pathogen. Most organisms have DNA that can be used as a source of primers of complementary DNA (cDNA). For viruses with RNA genomes it is necessary to synthesize cDNA copies of the genome using an enzyme called **reverse transcriptase (RT)** before the viral genome can be amplified by PCR. Then, through a repeated cycle of annealing the primers to the cDNA to create short, double-stranded DNA segments, extension of these double-stranded segments by the enzyme *Taq* polymerase, and melting to separate the double strands, the concentration of pathogen DNA can be multiplied over a million-fold to readily detectable concentrations (Figure 6).

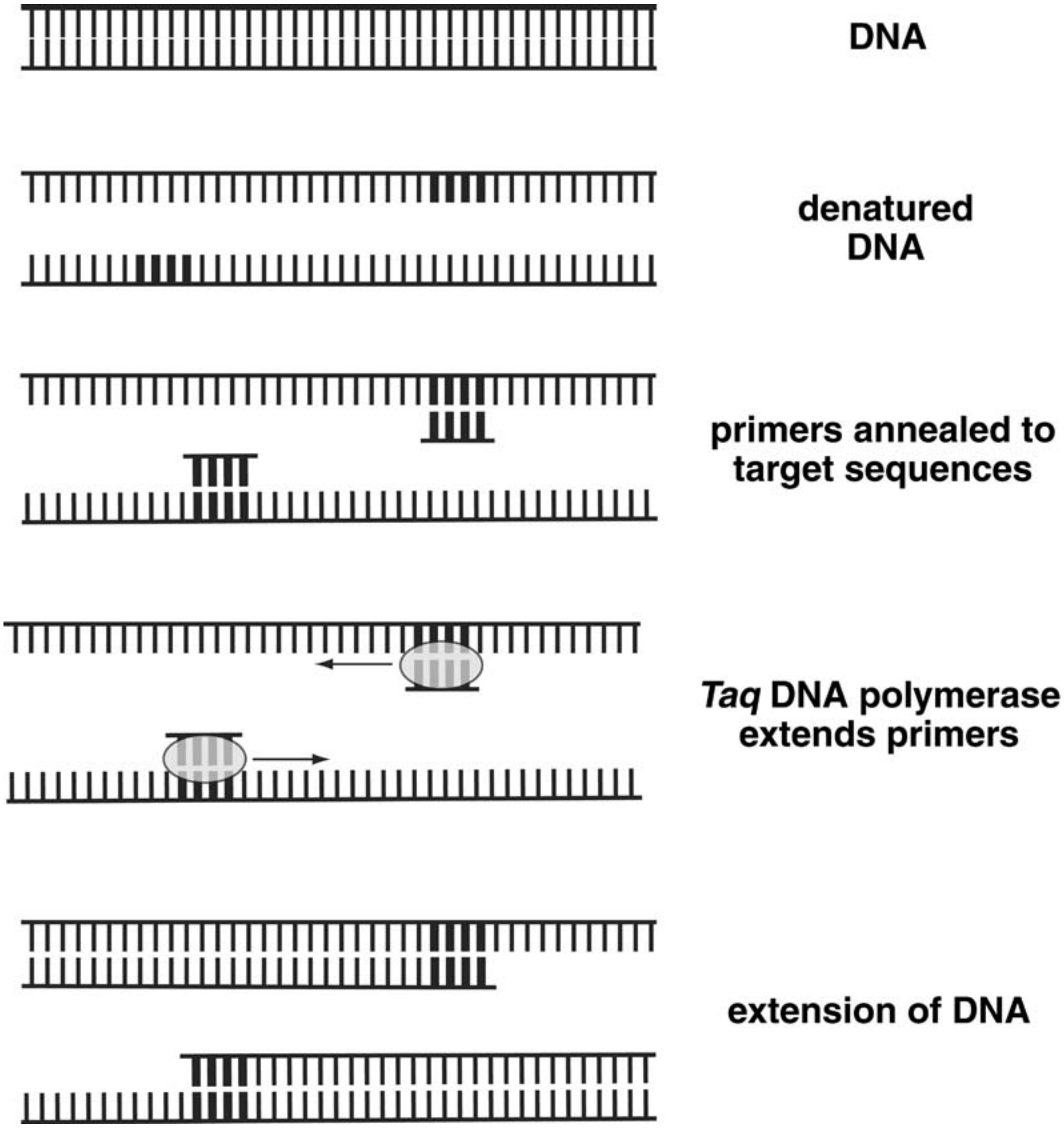
DNA
denatured DNA
primers annealed to target sequences
Taq DNA polymerase extends primers
extension of DNA

Figure 5. Diagram of first steps of polymerase chain reaction (PCR).

Figure 6. A scientist using a polymerase chain reaction (PCR) machine.

Like ELISA, the specificity of PCR varies, depending on the uniqueness of the genome sections selected for amplification. PCR has advantages in that the assay is more sensitive than ELISA (can detect lower populations of pathogen). In addition, primers can be designed to detect a single pathogen or a single race of the pathogen. For diagnostic purposes, a general primer from a pathogen might be more useful, whereas a more specific primer pair should be chosen for detection of new races of a pathogen in research or for quarantine purposes.

PCR tests require specialized equipment, which is relatively expensive. However, some equipment that can be used in the field has been developed, and new technologies (realtime PCR) have shortened the time required to complete the test to less than a day.

Polymerase Chain Reaction (PCR)

- **requires primers specific to the pathogen to be detected**
- **can be used for general or specific pathogen assays**
- **requires different primers for each pathogen**
- **can be more sensitive than ELISA**
- **requires specialized equipment to complete several cycles of DNA copying for detection**
- **usually requires that a plant sample be sent to a specialized lab for diagnosis**

Online resource for PCR

Dolan DNA Learning Center, Coldspring Harbor Laboratory, PCR animation

Which to use: Immunoassay, PCR, or standard microscopic examination and culturing?

The choice of method used to detect a plant pathogen often depends on the goals of the program or the study. If the goal of a certification program is to select only plants that are pathogen-free, then sensitivity is the prime criterion, and a test such as PCR may be used. If a very large number of samples must be processed, for example in a large epidemiological study, then rapidity and cost-effectiveness are key considerations, and ELISA may be the method of choice.

Diagnostic laboratories may stock primers and antisera of common and important pathogens and then send plant material to specialty laboratories for more complete testing as needed. Culturing and bioassays are still often used as the first steps to determine what pathogen is causing a disease problem. This is done for easily identified fungi and bacteria. Some nematodes can be readily extracted from plant materials and identified. Even though standard diagnostic methods may take days or weeks to complete, they tend to be inexpensive and can be accomplished without specialized equipment. These methods also are useful for detection of pathogens for which antisera and primers are not yet available or for diagnosis of new or unknown pathogens. For these reasons, no single method is universally used for plant disease diagnosis.

Diagnostic Resources

Books

American Phytopathological Society. Plant Disease Compendium Series. APS, St. Paul, MN. More than 35 volumes, several available in Spanish, describing diseases of a wide variety of fruit, vegetable, and field crops and turf, flower, and landscape plants, with many color illustrations. A complete list of titles in this series is available at the APS website.

Chase, A. R. 1997. Foliage Plant Diseases: Diagnosis and Control. American Phytopathological Society Press, St. Paul, MN.

Dhingra, O. D., and Sinclair, J. B. 1985. Basic Plant Pathology Methods. CRC Press, Boca Raton, FL.

Dreistadt, S. H. 2001. IPM for Floriculture and Nurseries. Statewide IPM Program, Agriculture and Natural Resources, University of California, Oakland.

Fox, R. T. V. 1993. Principles of Diagnostic Techniques in Plant Pathology. International Mycological Institute. CAB International, Surrey, UK.

Gleason, M. L., Daughtrey, M. L., Chase, A. R., Moorman, G. W., and Mueller, D. S. 2009. Diseases of Herbaceous Perennials. American Phytopathological Society, St. Paul, MN.

Horst, R. K. 2001. Wescott's Plant Disease Handbook. 6th ed. Kluwer Academic Publishers, Boston, MA.

Jones, R. K., and Benson, D. M., eds. 2001. Diseases of Woody Ornamentals and Trees in Nurseries. American Phytopathological Society Press, St. Paul, MN.

Pirone, P. P. 1978. Diseases and Pests of Ornamental Plants. 5th ed. John Wiley and Sons, New York.

Sherf, A. F., and Macnab, A. A. 1986. Vegetable Diseases and Their Control. 2nd ed. John Wiley and Sons, New York.

Shurtleff, M. C., and Averre, C. W. III. 2000. Diagnosing Plant Diseases Caused by Nematodes. American Phytopathological Society, St. Paul, MN.

Shurtleff, M. C. and Averre, C. W. III. 1997. The Plant Disease Clinic and Field Diagnosis of Abiotic Diseases. American Phytopathological Society, St. Paul, MN.

Sinclair, W. A., and Lyon, H. H. 2005. Diseases of Trees and Shrubs. 2nd ed. Cornell University Press, Ithaca, NY.

Waller, J. M., Ritchie, B. J., and Holderness, M. 1998. Plant Clinic Handbook. International Mycological Institute Technical Handbooks No. 3. CAB International, Surrey, UK.

Diagnostic services

Each state in the United States provides diagnostic services through the land-grant university extension programs. These can usually be found with a simple Internet search.

Some companies that provide immunoassay- and DNA-based diagnostic screening and/or kits for plant diseases

Acris Antibodies-Origene (Germany)
Agdia, Inc. (United States)
BIOREBA AG (Switzerland)/Eurofins (United States)
DSMZ (Germany)
Envirologix (United States)
Pocket Diagnostics (United Kingdom)

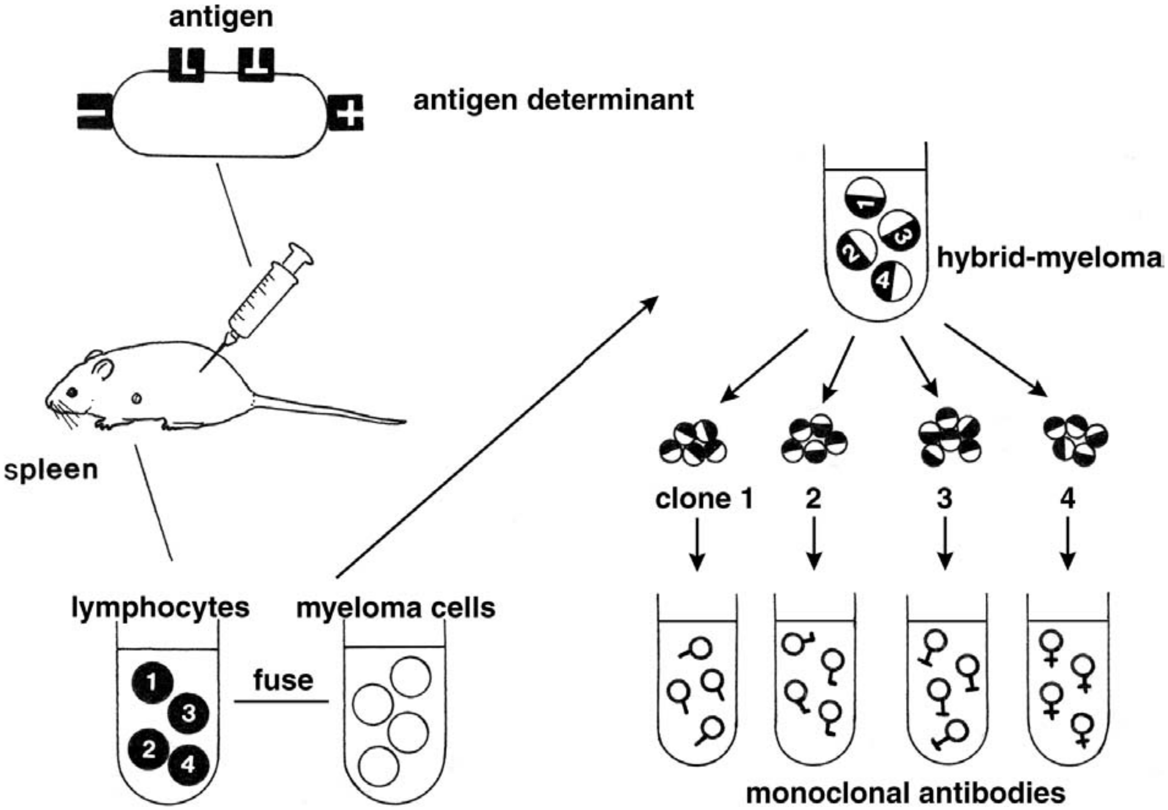
antigen
antigen determinant
spleen
lymphocytes
myeloma cells
fuse
1
2
3
4
hybrid-myeloma
clone 1
2
3
4
monoclonal antibodies

Figure 1.
Steps in the process of generating monoclonal antibodies.

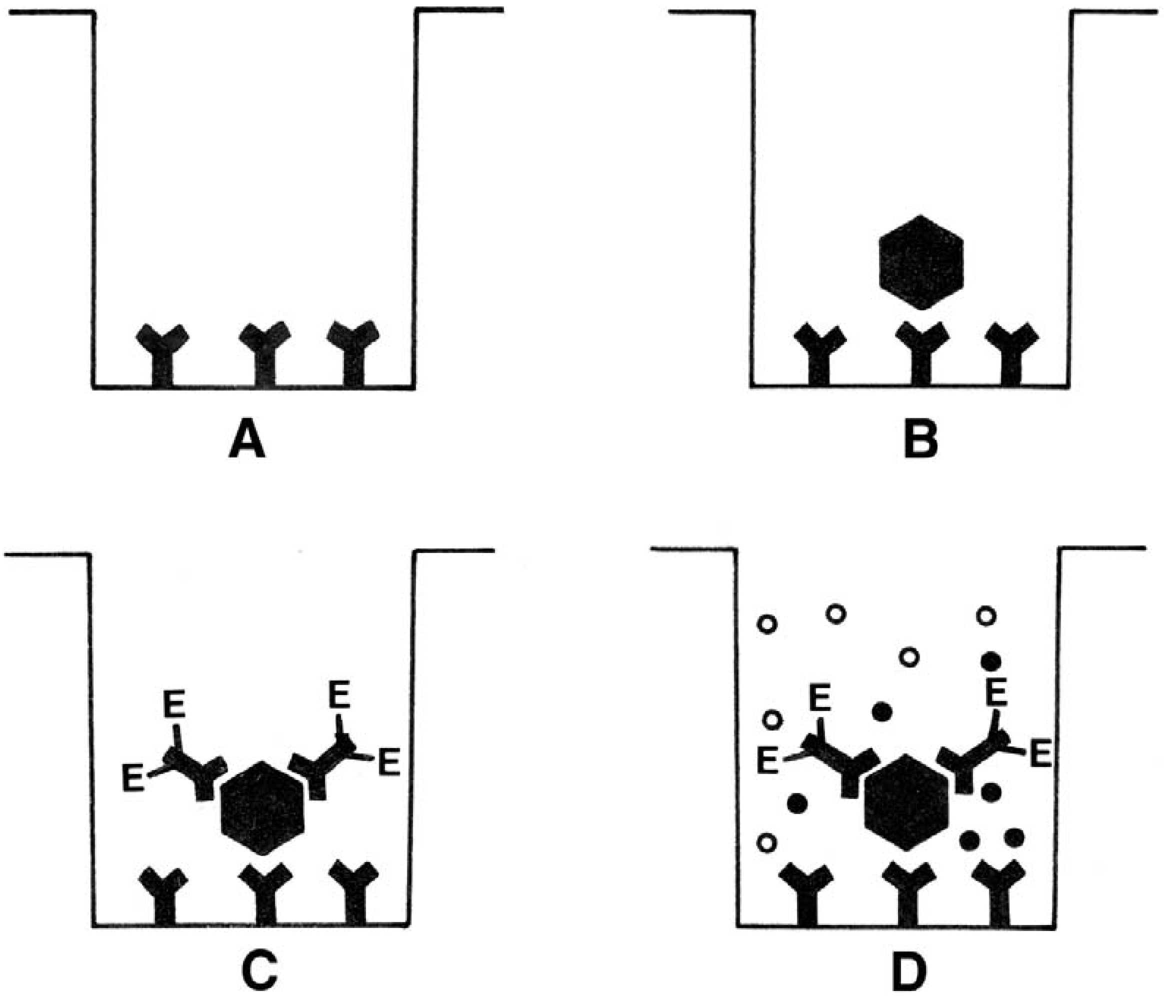

Figure 2.
Procedures used for enzyme-linked immunosorbent assay (ELISA). **A,** A specific antibody is adsorbed to the plate, which is incubated and then washed to remove unbound materials. **B,** The test sample containing the pathogen (antigen) is added, and the plate is incubated and washed. **C,** The enzyme-labeled (E) specific antibody (conjugate) is added, and the plate is incubated and washed. **D,** The enzyme substrate (circles) is added and is acted upon by the enzyme to produce the colored product (dark circles).

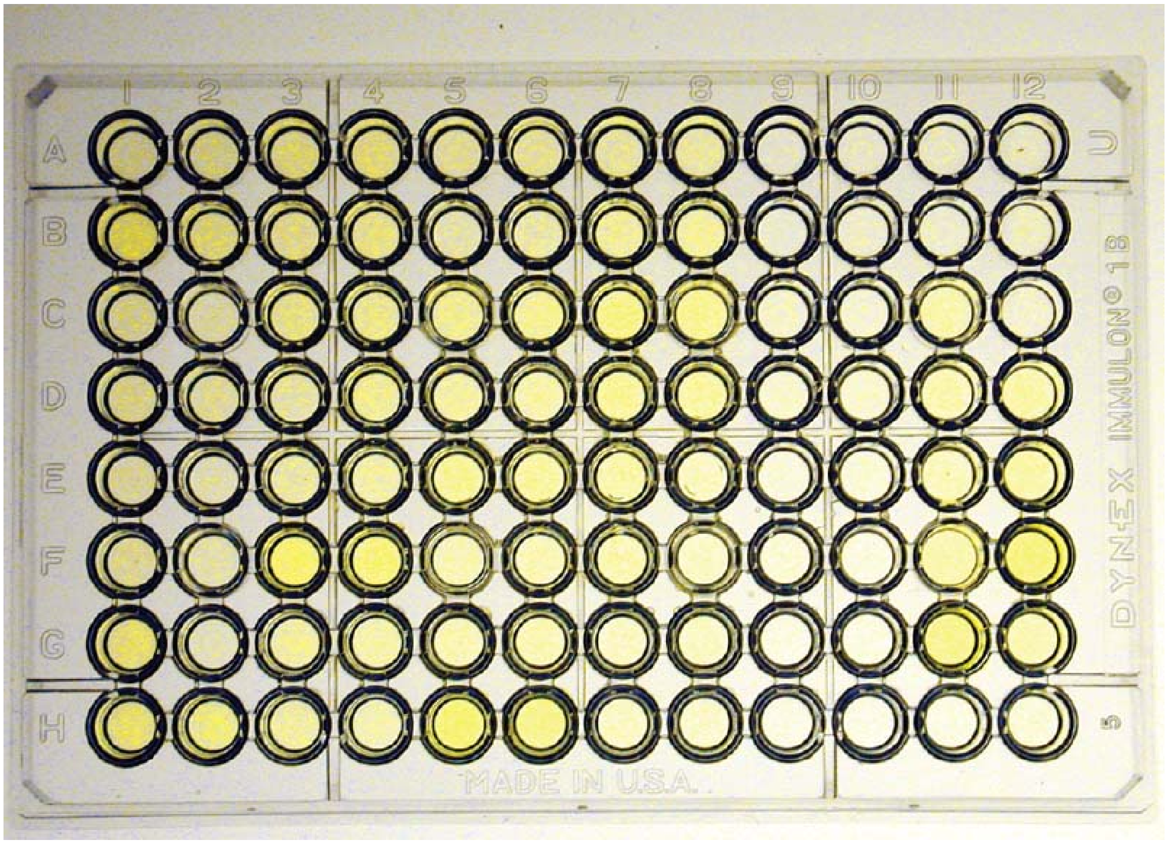
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Figure 3.

Multiwell plate for ELISA. Each well contains a test sample. The wells showing yellow color are positive.

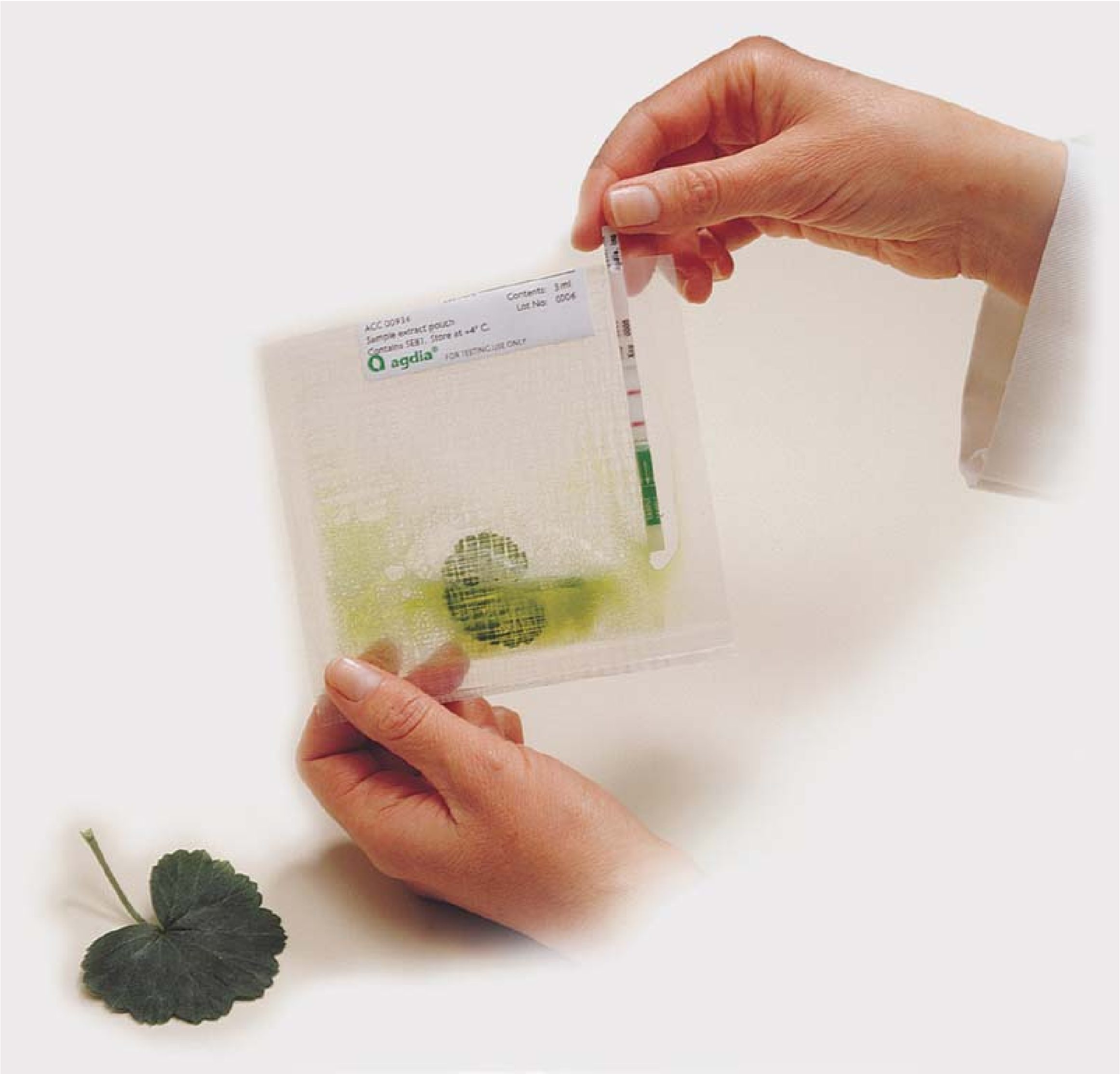

Figure 4.
Immunostrips for rapid on-site pathogen identification using ELISA. Color bands form if the pathogen is present in the sample.

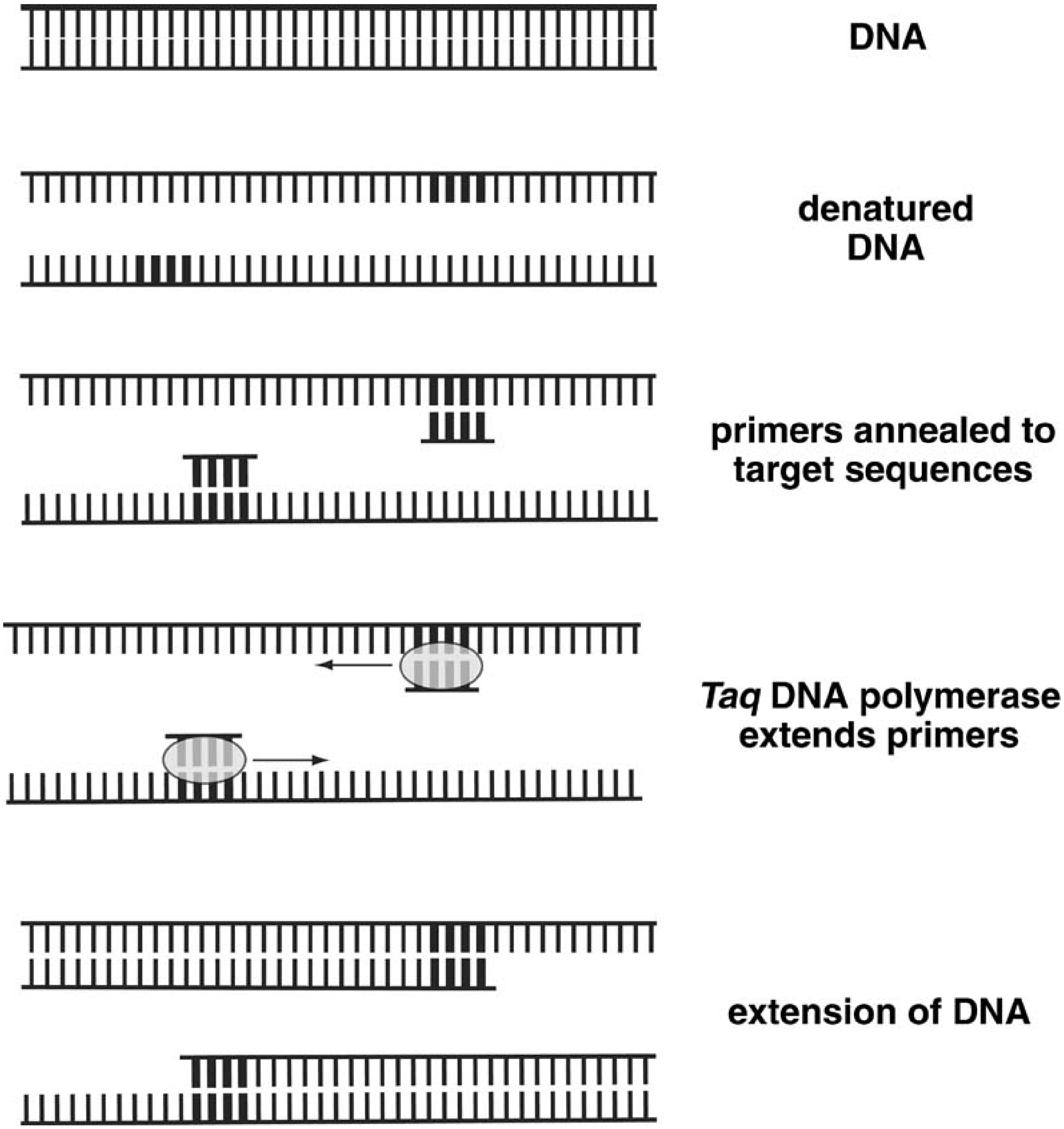
DNA
denatured DNA
primers annealed to target sequences
Taq DNA polymerase extends primers
extension of DNA

Figure 5.
Diagram of first steps of polymerase chain reaction (PCR).

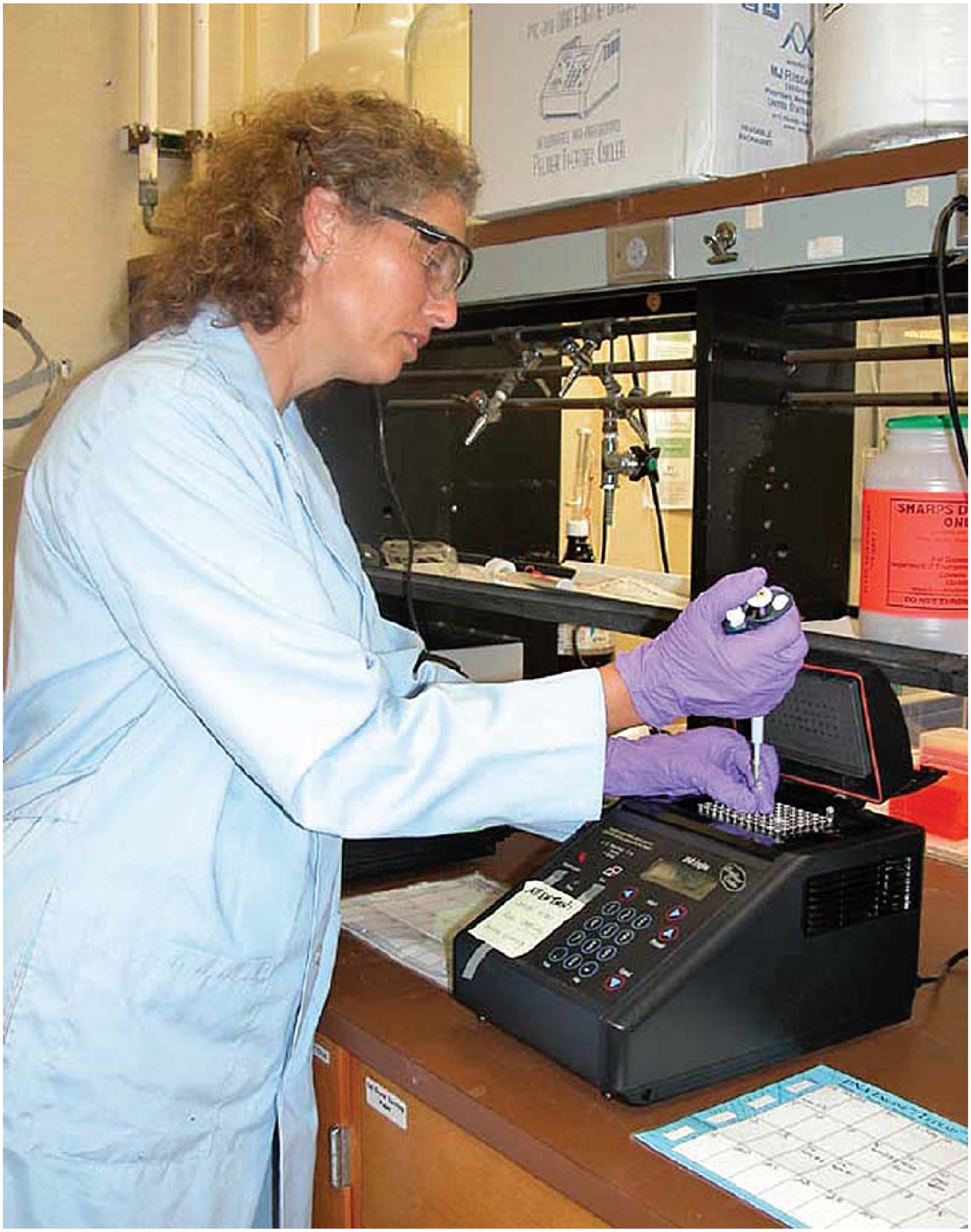

Figure 6.
A scientist using a polymerase chain reaction (PCR) machine.

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Glossary

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abaxial directed away from the stem of a plant; pertaining to the lower surface of a leaf (contrasts with adaxial)

abiotic pertaining to the absence of life Abiotic diseases are not caused by living organisms, but by chemical and physical factors. (see also noninfectious) (contrasts with biotic, infectious)

abscise to separate from a plant, as leaves, flowers, and fruits do when they fall

abscission the shedding of leaves or other plant parts as the result of physical weakness in a specialized layer of cells (the abscission layer) that develops at the base of the structure

acceptable daily intake (acronym ADI) a measure of the level of a chemical, i.e., a pesticide residue, that is believed to be able to be consumed on a daily basis over a lifetime without harm; used by the U.S. government to establish safe levels of potentially hazardous substances in food

acervulus (pl. acervuli) erumpent, cushionlike fruiting body bearing conidiophores, conidia, and sometimes setae

acid having a pH less than 7 (contrasts with alkaline, base)

acid precipitation precipitation (fog, rain, snow) with a low pH, resulting from the presence of nitric and sulfuric acid formed by the reaction of air pollutants ($N0_x$ and $S0_2$) with water

acid rain precipitation with a low pH, resulting from the presence of nitric and sulfuric acid formed by the combination of air pollutants ($N0_x$ and $S0_2$) with water

acropetal upward from the base to the apex of a shoot of a plant; in fungi, the production of spores in succession in the direction of the apex so that the apical spore is the youngest (contrasts with basipetal)

actinomycete a group of microorganisms similar to bacteria that produce long filaments

acute pertaining to symptoms that develop suddenly (contrasts with chronic)

acute toxicity ability of a single dose of a compound to poison (contrasts with chronic toxicity)

adaxial directed toward the stem of a plant; pertaining to the upper surface of a leaf (contrasts with abaxial)

ADI (acronym for acceptable daily intake) a measure of the level of a chemical, i.e., a pesticide residue, that is believed to be able to be consumed on a daily basis over a lifetime without harm; used by the U.S. government to establish safe levels of potentially hazardous substances in food

adpressed (syn. appressed) closely flattened down or pressed against a surface

adventitious arising from other than the usual place, as roots from a stem rather than as branches of a root

aecial host the host plant on which a heteroecious rust fungus produces aecia and pycnia (contrasts with telial host)

aeciospore dikaryotic spore of a rust fungus produced in an aecium; in heteroecious rusts, a spore stage that infects the alternate host

aecium (pl. aecia; adj. aecial) the fruiting body of a rust fungus in which the first dikaryotic spores (aeciospores) are produced

aerial occurring in the air

aerification the act of infusing or forcing air into, for example, soil

aerobic living only in the presence of oxygen

aflatoxin chemical byproduct from *Aspergillus flavus* and *A. parasiticus* harmful to humans and other animals

agar jellylike material derived from algae and used to solidify liquid culture media; term also applied to the medium itself

aggressiveness relative ability of a plant pathogen to colonize and cause damage to plants (see also virulence)

air pollution any air contaminant that causes undesirable effects on living organisms or materials

alate winged (contrasts with apterous)

albino (n. albinism) white or light-colored; having a marked deficiency in pigmentation

alkaline having basic (nonacidic) properties; pH greater than 7 (see also base) (contrasts with acid)

alkaloid nitrogen-containing ring compound produced by plants that causes physiological effects in animals

allele any of one or more alternative forms of a gene

allelopathy (adj. allelopathic) ability of one species to inhibit or prevent the growth of another species through the production of toxic substance(s)

alternate host one of two kinds of plant required by a heteroecious rust fungus to complete its life cycle; sometimes used as a general term for the aecial host or for the economically less important host (see also aecial host, telial host)

alternative host a plant other than the main host that a parasite can colonize; alternative hosts are not required for completion of the developmental cycle of the parasite

alternation of generations a reproductive cycle in which a haploid phase alternates with a diploid phase

AM (acronym for arbuscular mycorrhiza) symbiotic association between a nonpathogenic or weakly pathogenic fungus and the roots of plants in which fungal hyphae invade cortical cells of the root and produce vesicles and arbuscles (see also endomycorrhiza) (contrasts with ectomycorrhiza)

amino acid organic nitrogenous acid from which protein molecules are constructed

amphid a chemosensory organ located in the anterior region of a nematode

amphigynous having an antheridium through which the oogonium grows, as in many *Phytophthora* species (contrasts with paragynous)

amphimixis copulation of two unrelated cells and nuclei, e.g., egg and sperm; reproduction by a sexual process

amphimobile able to move both upward and downward in phloem, as do some pesticides

anaerobic living in the absence of oxygen

anamorph (adj. anamorphic) the asexual form in the life cycle of a fungus, when asexual spores (such as conidia) or no spores are produced (see also imperfect state) (contrasts with holomorph, perfect state, teleomorph)

anastomosis (pl. anastomoses) fusion between branches of the same or different structures (e.g., hyphae) to make a network

annual a plant that completes its life cycle and dies within one year (contrasts with biennial, perennial)

annual ring single-year growth of xylem in a woody stem

annulation grooved band in the cuticle of some nematodes

antagonism a general term for interference between organisms that may include antibiosis or competition for nutrients or space; action of two or more pesticides that reduces the effectiveness of one or all (contrasts with synergism)

antagonist an organism or substance that limits or counteracts the action of another

anterior toward the front or head (contrasts with posterior)

anther pollen-bearing portion of a flower

antheridium (pl. antheridia) male sexual organ (male gametangium) found in some fungi

anthesis the period of the opening of a flower during which pollination can occur

anthracnose disease caused by acervuli-forming fungi (order Melanconiales) and characterized by dark, sunken lesions and necrosis

antibiosis an association between organisms, or between an organism and a metabolic product of another organism, that is harmful to one of them

antibiotic a chemical compound produced by one microorganism that inhibits growth of or kills other living organisms

antibody a specific protein formed in animals in response to the presence of an antigen

antigen any foreign chemical (normally a protein) that induces antibody formation in animals

antiserum (pl. antisera) blood serum containing antibodies

apex (pl. apices; adj. apical) tip of a root or shoot containing the apical meristem

aphid small, sucking insect of the family Aphididae (order Hemiptera) that produces honeydew and injures plants when in large populations

apothecium (pl. apothecia) open, cuplike or saucerlike, ascus-bearing fungal fruiting body (ascocarp), often supported on a stalk

appressed (syn. adpressed) closely flattened down or pressed against a surface

appressorium (pl. appressoria) swollen, flattened portion of a fungal filament that adheres to the surface of a higher plant, providing anchorage for invasion by a fungus

apterous wingless (contrasts with alate)

arable able to be cultivated for agriculture

arbuscular mycorrhiza (acronym AM) symbiotic association between a nonpathogenic or weakly pathogenic fungus and the roots of plants in which fungal hyphae invade cortical cells of the root and produce vesicles and arbuscles (see also endomycorrhiza) (contrasts with ectomycorrhiza)

arbuscule (adj. arbuscular) branched haustorial structure of certain endomycorrhizal fungi that forms within living cells of the root

area under disease progress curve (acronym AUDPC) a measure of the total amount of disease over a period of time, determined from graphs of disease vs. time, which can be used to compare epidemics quantitatively

arthropod member of the phylum Arthropoda, which consists of animals with articulated bodies and limbs and which includes insects, arachnids, and crustaceans

ascocarp sexual fruiting body of an ascomycetous fungus that produces asci and ascospores, e.g., apothecium, as-costroma, chasmothecium/cleistothecium, perithecium, pseudothecium (see also ascoma)

ascogenous pertaining to ascus-producing hyphae

ascogonium (pl. ascogonia) a specialized cell that gives rise to the hyphae that produce asci

ascoma (pl. ascomata) sexual fruiting body of an ascomycetous fungus that produces asci and ascospores, e.g., apothecium, ascostroma, chasmothecium/cleistothecium, perithecium, pseudothecium (see also ascocarp)

ascomycetes (adj. ascomycetous) a group of fungi, also called the Ascomycota, that produce sexual spores (ascospores) within a saclike structure called an ascus

ascospore sexual spore borne in an ascus

ascostroma (pl. ascostromata) a fruiting body containing bitunicate (double-walled) asci in locules (cavities); usually dark with multiple locules, but sometimes single (see also pseudothecium)

ascus (pl. asci) saclike structure containing ascospores (typically eight) and usually borne in a fungal fruiting body

aseptate having no cross walls; lacking septa (see also nonseptate, coenocytic)

asexual vegetative; without sex organs, gametes, or sexual spores; the imperfect or anamorphic stage of a fungus

asexual reproduction any type of reproduction not involving the union of gametes and meiosis (contrasts with sexual reproduction)

AUDPC (acronym for area under disease progress curve) a measure of the total amount of disease over a period of time, determined from graphs of disease vs. time, which can be used to compare epidemics quantitatively

autoecious in reference to rust fungi, producing all spore forms on one species of host plant (contrasts with heteroecious)

autotroph an organism that synthesizes its nutritive substances from inorganic molecules; e.g., plants capable of photosynthesis (contrasts with heterotroph)

auxin plant hormone (growth regulator) influencing growth through cell elongation

avirulence (*avr*) gene gene in a pathogen that causes the pathogen to elicit an incompatible (defense) response in a resistant host plant and may enhance pathogen virulence in a susceptible host plant. The outcome of the interaction of an avirulence gene product with its corresponding plant resistance (R) gene product is usually a hypersensitive response.

avirulent unable to cause disease (see also nonpathogenic) (contrasts with virulent)

avoidance principle of plant disease management in which plants are grown at times or locations where the pathogen is inactive or not present

awn bristlelike structure at the apex of the outer bract of some cereal and grass flowers

axenic pertaining to a culture grown in the absence of living bacteria or other organisms; pure culture

axil the angle formed by the leaf petiole and the stem

axillary pertaining to or placed within an axil

axillary bud bud that develops in the axil of a leaf (see also lateral bud)

bacilliform shaped like short rods with rounded ends

backcross to cross (mate) an offspring with one of its parents

bacterial streaming large populations of bacteria that exude from the cut surface of infected plant tissue, observable with a microscope

bactericide a chemical or physical agent that kills bacteria

bacteriocin a protein antibiotic, one or more types of which can be produced and excreted by certain strains of bacteria

bacteriophage a virus that infects a bacterium

bacterium (pl. bacteria) a prokaryotic, microscopic, single-celled organism with a cell wall that increases by binary fission

bark all tissues outside the vascular cambium of a woody plant, generally including the cork layers, cork cambium (phellogen), and phloem

basal knob structure at the base of a nematode stylet (see also stylet knob)

base having a pH greater than 7 (see also alkaline) (contrasts with acid)

basidiocarp sexual fruiting body of a basidiomycetous fungus (see also basidioma)

basidioma (pl. basidiomata) sexual fruiting body of a basidiomycetous fungus (see also basidiocarp)

basidiomycetes (adj. basidiomycetous) a fungus group, also called the Basidiomycota, characterized by the formation of external basidiospores on basidia

basidiospore haploid (1N) sexual spore produced on a basidium

basidium (pl. basidia; adj. basidial) specialized cell or organ, often club-shaped, in which karyogamy and meiosis occur, followed by production of externally borne basidiospores (generally four) that are haploid. There are several types of basidia.

basipetal downward from the apex toward the base of a shoot; referring to development in the direction of the base so that the apical part is oldest (contrasts with acropetal)

beetle any insect of the order Coleoptera characterized by elytra (thickened outer wings), chewing mouth parts, and complete metamorphosis

bicellular two-celled

biennial a plant that produces seed and dies at the end of its second year of growth (contrasts with annual, perennial)

biflagellate having two flagella

binary fission a type of asexual reproduction in which two cells, usually of similar size and shape, are formed by the growth and division of one cell

binomial, Latin the scientific name of an organism, composed of two names, the first designating the genus and second the specific epithet, together making the species name

binucleate having two nuclei

bioassay any test (assay) using a living organism

biocide a compound toxic to all forms of life

biocontrol exploitation by humans of the natural competition, parasitism, and/or antagonism of organisms for management of pests and pathogens (see also biological control)

biolistic transformation a method used for genetic engineering in which plant cells are bombarded with metal particles coated with foreign genes using a "gene gun" (see also gene gun)

biological control exploitation by humans of the natural competition, parasitism, and/or antagonism of organisms for management of pests and pathogens (see also biocontrol)

biotechnology the development of genetically modified organisms through the use of modern technology and processes, including genetic engineering

biotic relating to life, as disease caused by living organisms (see also infectious)(contrasts with abiotic, noninfectious)

biotroph an organism that can live and multiply only on another living organism (see also obligate parasite) (contrasts with necrotroph)

biotype a subdivision of a species, subspecies, or race based on some identifiable physiological trait such as a specific virulence pattern

bitunicate having a double ascus wall (contrasts with unitunicate)

blasting a symptom of plant disease characterized by shedding of unopened buds; classically, the failure to produce fruit or seed

bleeding sap flow from a wound

blight sudden, severe, and extensive spotting, discoloration, wilting, or destruction of leaves, flowers, stems, or entire plants

blotch necrotic area of tissue irregular in form

borer insect or insect larva that forms tunnels or cavities in the bark or within the wood of trees

boundary layer the calm layer of air on surfaces below the layer of more turbulent air

bract a reduced leaf associated with a flower or inflorescence; modified leaf from the axil from which a flower arises

breaking disease symptom, usually caused by a virus, involving addition or loss of flower color to create a variegated pattern (see also flower break)

breeding line plant strain used in a plant-breeding program and usually containing one or more desirable agronomic or breeding characteristics

broadcast application application by spreading or scattering on the soil surface

brooming profuse branching of woody stems from a single stem position

brown rot (of wood) wood decay resulting from selective removal of cellulose and hemicellulose, leaving a brown amorphous residue that usually cracks into cubical blocks and consists largely of slightly modified lignin

bud a terminal or axillary structure on a stem consisting of a small mass of meristematic tissue, generally covered wholly or in part by modified scale leaves

budding a special type of plant grafting using a single bud on the rootstock of another plant; method of asexual reproduction in fungi, such as yeasts

bud scale a specialized protective leaf of a bud

bud wood wood consisting of strong, young shoots bearing buds suitable for use in budding

bug any insect of the order Hemiptera characterized in part by piercing-sucking mouth parts, a triangular scutellum, two pairs of wings, and gradual metamorphosis

bulb a short, flattened, usually globose or disk-shaped, underground, perennial, storage organ composed of concentric layers of overlapping fleshy scale leaves attached to a stem plate at the base; essentially a subterranean bud

bunt ball smut sorus, filled with teliospores, that replaces a cereal or grass kernel but is covered by plant tissue at maturity

burr knot a rough outgrowth, often present on the trunks or roots of certain trees

bursa extension or flap of cuticle at the side of the male nematode sex organ, used for orienting during mating

bush-type pertaining to a plant with determinate growth

callose amorphous, hardened, carbohydrate constituent of plant cell walls, commonly developing upon injury

callus specialized tissues that form over a wound or cut in a plant; cork cambium may form and the cells produced will gradually seal the wound

calyx outermost flower whorl; sepals, collectively

canker a plant disease characterized (in woody plants) by the death of cambium tissue and loss and/or malformation of bark, or (in nonwoody plants) by the formation of sharply delineated, dry, necrotic, localized lesions on the stem; "canker" may also be used to refer to the lesion itself, particularly in woody plants

canopy the expanded leafy top of a plant or plants

capsid the protective layer of protein surrounding the nucleic acid core of a virus; the protein molecules that make up this layer (see also coat protein)

capsule gel-like material surrounding a bacterial cell

carbohydrate any of various chemical compounds composed of carbon, hydrogen, and oxygen, such as sugars, starches, and cellulose

carbon cycle the continuous circulation of carbon atoms from inorganic carbon dioxide (CO_2) to organic molecules and back to CO_2

carcinogen a substance or agent that causes cancer

carpel the ovule-bearing structure of a flower in angiosperms

casting premature loss of abscised leaves or twigs

catkin conelike cluster of male or female flowers; conelike fruit of angiosperms

causal agent organism or agent that incites and governs disease or injury

cell membrane structure that bounds a cell and helps control the movement of substances into and out of the cell

cell wall protective, resistant, but permeable structure secreted externally to the cell membrane in plants, bacteria, fungi, and certain other organisms

cellulose carbohydrate polysaccharide composed of glucose units; major component of plant primary cell wall

center of origin geographical area where a plant originated

centromere the constricted portion of a chromosome to which, in mitosis, the chromosomal fiber is attached

certification program by which seeds, propagative plant material, or nursery stock are produced and sold under inspection by an official agency to maintain genetic identity and purity and freedom from harmful pathogens, insect pests, and weed seeds

cfu (acronym for colony-forming unit); the number of colonies formed per unit of volume or weight of a cell or spore suspension

chasmothecium the ascocarp of a powdery mildew fungus; it has no natural opening at maturity, but opens by the rupturing of its wall (see also cleistothecium)

chemotaxis (syn. chemotropism) movement or growth of an organism in response to changing concentration of a chemical stimulus, often in relation to food or for mating

chemotherapy treatment of plant disease with chemicals (e.g., antibiotics or fungicides) absorbed and translocated internally

chimera (or chimaera) plant or organ consisting of two or more genetically different tissues

chitin complex polysaccharide carbohydrate in fungal cell walls, animal exoskeletons, and nematode egg shells

chlamydospore thick-walled or double-walled asexual resting spore formed from hyphal cells (terminal or intercalary) or by transformation of conidial cells that can function as an overwintering stage

chlorophyll (adj. chlorophyllous) one of a group of green pigments found in chloroplasts and important in photosynthesis

chloroplast disklike structure containing chlorophyll in which photosynthesis occurs in green plants

chlorosis (adj. chlorotic) failure of chlorophyll development, caused by disease or a nutritional disturbance; fading of green plant color to light green, yellow, or white

chromosome the structure that contains the genes of an organism; in eukaryotes, chromosomes are in the nucleus and can be visualized with an optical microscope as threads or rods during meiosis and mitosis; in bacteria, the chromosome is usually a single circle of DNA that cannot be visualized with an optical microscope

chronic pertaining to slow-developing, persistent, or recurring symptoms (contrasts with acute)

chronic toxicity poisoning resulting from exposure to low levels of a compound over a period of time (contrasts with acute toxicity)

chytridiomycetes a fungus group, also called the Chytridiomycota, characterized by the production of motile zoospores and resting sporangia; the plant-pathogenic species are all biotrophs that are restricted to the cells of their host

circulative transmission virus transmission characterized by a long period of acquisition of the virus by a vector (typically an insect), a latent period of several hours before the vector is able to transmit the virus, and retention of the virus by the vector for a long period, usually several days; the virus circulates in the body of the vector (see also persistent transmission, propagative transmission) (contrasts with nonpersistent transmission, stylet-borne transmission)

cirrhus (pl. cirrhi) a curled, tendril-like mass of exuded spores, held together by a slimy matrix

clamp connection a bridge- or buckle-hyphal protrusion in basidiomycetous fungi, formed at cell division and connecting the newly divided cells

clavate (or claviform) club-shaped

cleistothecium (pl. cleistothecia) a spherical ascocarp that is closed at maturity (see also chasmothecium)

clone (n.) a population of cells or organisms of identical genotype; a population of recombinant DNA molecules all carrying the same inserted sequence; (v.) to vegetatively propagate an organism to produce a population of identical individuals; to use in vitro recombination techniques to insert a particular DNA sequence into a vector

coalesce to grow together into one body or spot

coat protein the protective layer of protein surrounding the nucleic acid core of a virus; the protein molecules that make up this layer (see also capsid)

coccus (pl. cocci) a spherical (or near-spherical) bacterial cell

codon a particular sequence of three nucleotides in a nucleic acid that codes for a specific amino acid or termination of a polypeptide chain (see also triplet codon)

coelomycetes archaic term for a group of fungi in the deuteromycetes (imperfect fungi, Fungi Imperfecti) that produce pycnidia or acervuli (contrasts with hyphomycetes)

coenocytic having multiple nuclei embedded in cytoplasm without cross walls (see also aseptate, nonseptate)

collar the portion of the seedling or plant near the surface of the soil; in grafted woody plants, the scion portion of the plant near the soil surface

collenchyma supporting tissue in soft stems and other plant parts, composed of elongated living parenchyma cells with unevenly thickened primary walls, often bordering veins in dicot leaves; the “strings” in celery

colonization establishment and ramification of a pathogen within a host plant

colonize to infect and ramify through plant tissue via the growth of a pathogen

colony a growth of a microorganism in mass, especially as a pure culture

colony-forming unit (acronym cfu) the number of colonies formed per unit of volume or weight of a cell or spore suspension

companion cell specialized cell adjacent to sieve tubes in phloem

compartmentalization isolation of a specific tissue area by host barrier tissues

complete resistance a resistant reaction in which some aspect of disease development, usually symptom expression or pathogen reproduction, is completely stopped (contrasts with partial resistance)

compost a mixture of organic residues and soil that is allowed to decompose biologically

concentric having one circle within another with a common center

conidiogenesis the formation of asexual spores (conidia)

conidiogenous producing and bearing conidia

conidioma (pl. conidiomata) specialized conidia-bearing structure, e.g., acervulus, pycnidium, sporodochium, synnema

conidiophore simple or branched hypha on which conidia are produced

conidium (pl. conidia) an asexual, nonmotile fungal spore that develops externally or is liberated from the cell that formed it

conjugation the temporary contact of bacterial cells during which genetic material is transferred

conk a shelflike, typically hardened basidiocarp of a wood-decaying fungus, usually a polypore

constitutive present or produced under all conditions (contrasts with induced)

contact fungicide a fungicide that remains on the surface where it is applied; no after-infection activity (see also protectant fungicide) (contrasts with systemic fungicide)

continuous cropping growing the same crop in the same location repeatedly

coppice a regrowth of woody plants by sprouts from stumps or roots

coremium (pl. coremia) a headlike cluster formed by compact or fused, generally upright conidiophores, with branches and spores (see also synnema)

cork external protective tissue of a stem or root, impermeable to water and gasses; the primary component of bark

corolla petals, collectively

cortex (adj. cortical) region of parenchyma tissue between the epidermis and the phloem in stems and roots; region beneath the rind of a sclerotium

cotyledon seed leaf, one in moncots and two in dicots; primary embryonic leaf within the seed in which nutrients for the new plant are stored

crop rotation the successive planting of different crop species; often used to improve soil fertility or to reduce disease and pest problems

cross-fertilization a process in which sexual reproduction occurs as a result of the fusion of sex cells from different individuals (contrasts with self-fertilization)

cross-pollination transfer of pollen from the anthers to the stigma of another flower (contrasts with self-pollination)

cross-protection the process whereby a normally susceptible host is infected with a less virulent pathogen (usually a virus) and thereby becomes resistant to infection by a second, usually related, more virulent pathogen

crown upper dome of a tree, bearing leaves, flowers, and fruits; junction of root and stem of a plant, usually at the soil line; in grafted woody plants, the rootstock portion of the plant near the soil surface

crozier the hook of an ascogenous hypha before ascus development; the curved apical portion of a blighted stem (see also shepherd's crook)

cryptobiosis (hidden life) a state of suspended metabolic activity during unfavorable environmental conditions (drying, cold); survival strategy of some plant-pathogenic nematodes

culm stem of grasses, cereals, and bamboos

curl distoration, puffing, and crinkling of a leaf resulting from unequal growth

cultivar (abbrev. cv.) a plant type within a species, resulting from deliberate genetic manipulation, which has recognizable characteristics (color, shape of flowers, fruits, seeds, and height or form) (see also variety)

cultural practices the manner in which plants are grown, including factors such as application of nutrients, irrigation practices, type of cultivation; may be used for disease management

culture growth and propagation of microorganisms on nutrient media; growth and propagation of living plants

cuticle (adj. cuticular) noncellular outer layer of an insect or a nematode; water-repellent, waxy layer of epidermal cells of plant parts, such as leaves, stems, and fruit

cv. (abbrev. for cultivar) a plant type within a species, resulting from deliberate genetic manipulation, which has recognizable characteristics (color, shape of flowers, fruits, seeds, and height or form) (see also variety)

cyst in fungi, a resting structure in a protective membrane or shell-like enclosure; in nematode females, the egg-laden carcass of a female nematode; in bacteria, a specialized type of bacterial cell enclosed in a thick wall, often dormant and resistant to environmental conditions

cytokinin plant hormone (growth regulator) that controls cell division and is important for shoot stimulation of callus in tissue culture

cytopathology the study of changes induced by disease at the cellular level

cytoplasm living protoplasm in a cell, except the nucleus

cytoplasmic inheritance inheritance of genes not located in the nucleus, i.e., those in mitochondria and chloroplasts (see also extrachromosomal inheritance, maternal inheritance)

damping-off death of a seedling before or shortly after emergence resulting from decomposition of the root and/or lower stem; it is common to distinguish between preemergence damping-off and postemergence damping-off

days to harvest the prescribed minimum number of days required by U.S. government regulations between a pesticide application and the harvest of the crop

decay the gradual decomposition of organic matter

deciduous describing a tree that sheds its leaves completely at the end of its annual growth period

decoy crop crop that stimulates germination of seeds of a parasitic plant such as witchweed (*Striga* spp.), but is not susceptible to infection by the parasitic plant; helps reduce seed populations of the parasite in soil so a susceptible crop can be planted

defoliation loss of leaves from a plant, whether normal or premature

degree-day the departure of the average daily temperature from a defined base (e.g., the minimum recognized temperature for the growth of a plant species); the number of degree-days may be totaled to assess the accumulated warmth of a particular year’s growing season

dehiscent opening when mature by an opening or by breaking into parts, as in anthers, asci, fruiting bodies, fruits, sporangia, etc. (contrasts with indehiscent)

demicyclic pertaining to a rust fungus that lacks the urediniospore (repeating) stage (e.g., many species of *Gymnosporangium*) (contrasts with macrocyclic, microcyclic)

dentate toothed

deoxyribonucleic acid (abbrev. DNA) the double-stranded, helical molecule that contains genetic code information; each repeating unit, or nucleotide, is composed of deoxyribose (a sugar), a phosphate group, and a purine (adenine or guanine) or a pyrimidine (thymine or cytosine) base

desiccate to dry out

determinate ceasing vegetative growth when the first flower or reproductive structure forms (contrasts with indeterminate)

deuteromycetes archaic term for a group of fungi without a sexual stage; the asexual stage of ascomycetes and basidiomycetes (see also Fungi Imperfecti, imperfect fungi)

diagnostic (n. diagnosis) pertaining to a distinguishing characteristic important for the identification of a disease or other condition

diapause a period of spontaneous dormancy, independent of environmental conditions, interrupting developmental activity in an embryo, larva, or pupa

dichotomous branching, often successively, into two, more-or-less equal arms

dicot (syn. dicotyledon) plant with two cotyledons or seed leaves (contrasts with monocot)

dieback (v. die back) progressive death of shoots, leaves, or roots, beginning at the tips

differential host (syn. differential cultivar) a plant host that on the basis of disease symptoms serves to distinguish between various strains or races of a given plant pathogen

differential medium culture medium that is used to distinguish between organisms that can grow on it

differentiation the physiological and morphological changes that occur in a cell, tissue, or organ during development from a juvenile state to a mature state

digitate having lobes radiating from a common center

dikaryotic (n. dikaryon) having two sexually compatible haploid nuclei per cell that divide simultaneously; this phase is called the dikaryophase (contrasts with haploid, diploid, polyploid)

dilution plating a method to obtain pure colonies of bacteria and fungi in which infected plant material or infested soil is diluted in sterilized water. Small samples of the water are spread on the medium surface of several petri plates to find which dilution will produce pure colonies.

dilution streaking repeated streaking of bacteria on the surface of a nutrient medium with a sterile metal loop to allow pure colonies to grow

dimorphic having two distinct shapes or forms

dioecious having male and female organs on separate and distinct individuals (used primarily for plants) (contrasts with monoecious)

diploid having two complete sets of chromosomes (2N chromosomes) (contrasts with haploid, dikaryotic, polyploid)

direct penetration penetration of plant tissues by a pathogen through barriers such as leaf cuticle by chemical and physical means (e.g., penetration peg) (contrasts with indirect penetration)

discomycetes a group of the ascomycetes in which the hymenium is exposed at maturity; one in which the fruiting body is an apothecium or discocarp

disease abnormal functioning of an organism

disease cycle succession of all events and interactions among the host, parasite, and environment that occur in a disease, from initial infection of the plant by a causal agent, through pathogenesis, to over-seasoning, until another infection occurs

disease incidence number of plants affected by a disease within a population

disease progress curve graph of some measure of disease (i.e., severity) over time

disease pyramid a memory aid similar to the disease triangle but including, in addition, the factor of time in the development of a disease (contrasts with disease triangle)

disease severity the measure of damage done by a disease

disease triangle a memory aid that diagrams the three important components necessary for disease: susceptible plant, virulent pathogen, and favorable environment (contrasts with disease pyramid)

disinfect to eliminate a pathogen from infected plant tissues

disinfest to kill pathogens that have not yet initiated disease, or other contaminating microorganisms, that occur in or on inanimate objects as such soil or tools, or that occur on the surface of plant parts such as seed

dispersal spread of infectious material (inoculum) from diseased plants to healthy plants (see also dissemination)

dissemination spread of infectious material (inoculum) from diseased plants to healthy plants (see also dispersal)

distal far from the point of attachment or origin; in a direction away from main body (contrasts with proximal)

DNA (acronym for deoxyribonucleic acid) the double-stranded, helical molecule that contains genetic code information; each repeating unit, or nucleotide, is composed of deoxyribose (a sugar), a phosphate group, and a purine (adenine or guanine) or a pyrimidine (thymine or cytosine) base

dodder parasitic seed plant (*Cuscuta* spp.) without leaves; a yellow filamentous vine

dolipore septum cross wall found in basidiomycetes and characterized by special swellings and membranes in association with the septal pore

dominant describes a phenotypic trait that is expressed in hybrid progeny of diploid organisms even when contributed by only one of the parents (contrasts with recessive)

dormancy (adj. dormant) a condition of suspended growth and reduced metabolism of an organism, generally induced by internal factors or environmental conditions as a mechanism of survival

downy mildew a plant disease in which the pathogen appears as a downy growth on the host surface; caused by a member of the oomycetes

drift (of pesticides) movement of airborne particles of a spray, dust, or vapor away from the target area during or shortly after an application

drought insufficient soil water for normal plant growth

durable resistance resistance that remains effective during prolonged and widespread use in an environment favorable to disease (see also horizontal resistance, race-nonspecific resistance) (contrasts with specific resistance, vertical resistance)

dwarfing underdevelopment of a plant or plant organs, which may be caused by disease, inadequate nutrition, or unfavorable environmental conditions

echinulate having small spines projecting from cell walls

ecology the study of the interactions that occur between individual organisms, groups of organisms, and organisms and their environment

economic threshold the pathogen density at or above which the value of crop losses (in the absence of management efforts) would exceed the cost of management practices

ecosystem community of living things and their environment

ectomycorrhiza (pl. ectomycorrhizae) symbiotic association between a nonpathogenic or weakly pathogenic fungus and the roots of plants in which fungal hyphae grow between and external to root cells (contrasts with arbuscular mycorrhiza, endomycorrhiza)

ectoparasite parasite that feeds from the exterior of its host (contrasts with endoparasite)

ectotrophic pertaining to fungal development primarily over the root surface

edema (also oedema) blisters produced on leaves and other plant parts under conditions of high moisture and restricted transpiration (see also intumescence)

effector a pathogen molecule, usually a protein, that is translocated into host cells where it may act to directly manipulate host innate immunity

egg mass group of eggs held together by a gelatinous matrix

electron microscope microscope that uses a focused beam of electrons to produce a greatly enlarged image of minute objects, such as viruses, in the same way that light is used in a compound microscope

elicitor a molecule produced by the host (or pathogen) that induces a response by the pathogen (or host)

ELISA (acronym for enzyme-linked immunosorbent assay) a serological test in which the sensitivity of the reaction is increased by attaching an enzyme that produces a colored product to one of the reactants

embryo an organism in the early stages of development, such as a young plant in the seed or a nematode before hatching from the egg

emergence growth of the seedling shoot through the surface of the soil

enation abnormal outgrowth from the surface of a stem or leaf

encapsidate to cover virus nucleic acid with a protein coat

encyst to form a cyst or protective covering

endemic native to a particular place; pertaining to a low and steady level of natural disease occurrence

endocarp inner layer of a fruit wall

endoconidium (pl. endoconidia) a conidium produced inside a hypha or conidiophore

endodermis layer of cells within the root between the vascular tissue and the cortex

endogenous arising from the inside (contrasts with exogenous)

endomycorrhiza (pl. endomycorrhizae) symbiotic association between a nonpathogenic or weakly pathogenic fungus and the roots of plants in which fungal hyphae invade cortical cells of the root (see also arbuscular mycorrhiza) (contrasts with ectomycorrhiza)

endoparasite parasitic organism that lives and feeds from inside its host (contrasts with ectoparasite)

endophyte plant developing inside another organism; also used for endoparasitic fungi found in grass species

endoplasmic reticulum system of interconnected cytoplasmic membranes that transports materials within the cell

endosperm nutritive tissue formed within the embryo sac of seed plants

endospore resistant, thick-walled asexual spore formed within a bacterial or fungal cell

Environmental Protection Agency (acronym EPA) the federal agency responsible for controlling the various aspects of air, water, and soil pollution, including pesticide regulations

enzyme protein that catalyzes a specific biochemical reaction

enzyme-linked immunosorbent assay (acronym ELISA) a serological test in which the sensitivity of the reaction is increased by attaching an enzyme that produces a colored product to one of the reactants

EPA (acronym for Environmental Protection Agency) the federal agency responsible for controlling the various aspects of air, water, and soil pollution, including pesticide regulations

epicotyl portion of the stem of a plant embryo or seedling above the node where the cotyledons are attached (contrasts with hypocotyl)

epidemic an increase of disease in a population; a general and serious outbreak of disease (see also epiphytotic)

epidemiology (adj. epidemiologic) the study of factors influencing the initiation, development, and spread of infectious disease; the study of disease in populations of plants

epidermis (adj. epidermal) surface layer of cells of leaves and other plant parts

epinasty abnormal, downward curling of a leaf, leaf part, or stem

epiphytic living on the surface of plants, but not as a parasite

epiphytotic an epidemic in a plant population (see also epidemic)

epiphyllous on the upper surface of a leaf

epiphyte organism growing on a plant surface, but not as a parasite

epitope an amino acid (or other) sequence that effects formation of an antibody

EPS (acronym for extracellular polysaccharide) sugar polymer that aids in movement, helps prevent desiccation, and contributes to the slimy appearance of bacteria, fungi, and nematodes; and probably promotes colonization of plant tissues and disease development by bacteria

eradication management of plant disease by eliminating the pathogen after it is established or by eliminating the plants that carry the pathogen

ergot a disease of certain grasses and cereals, especially rye, caused by *Claviceps* spp.; a sclerotium, or resting structure, produced by *Claviceps* spp. and other closely related fungi in infected flowers of parasitized grain plants

ergotism a disease caused by ingestion of grain contaminated with alkaloids of ergot fungi, often called the holy fire or St. Anthony's fire in the Middle Ages

erumpent bursting or erupting through the substrate surface

escape failure of inherently susceptible plants to become diseased, even though disease is prevalent

ethylene a plant hormone influencing various aspects of vegetative growth, fruit ripening, abscission of plant parts, and the senescence of flowers

etiolation elongation of stems caused by, for example, reduced light intensities

etiology the study of the causes of diseases

eukaryote an organism containing a membrane-bound nucleus and other organelles, i.e., all higher plants, animals, fungi, and protists (contrasts with prokaryote)

exclusion management of disease by excluding the pathogen or infected plant material from crop production areas (e.g., by quarantines and embargoes)

exogenous originating from the outside (contrasts with endogenous)

extracellular outside a cell

extracellular polysaccharide (acronym EPS) sugar polymer that aids in movement, helps prevent desiccation, and contributes to the slimy appearance of bacteria, fungi, and nematodes; and probably promotes colonization of plant tissues and disease development by bacteria

extrachromosomal inheritance inheritance of genes not located in the nucleus, i.e., those in mitochondria and chloroplasts (see also cytoplasmic inheritance, maternal inheritance)

exudate liquid excreted or discharged from diseased tissues, from roots and leaves, or by fungi

f. sp. (abbrev. for *forma specialis*) a taxonomic group within a pathogenic species defined in terms of host range, i.e., members of different *formae speciales* infect different groups of plants

facultative capable of changing lifestyle, e.g., from saprophytic to parasitic or the reverse

facultative parasite organism that is normally saprophytic but is capable of being parasitic

facultative saprophyte organism that is normally parasitic but is capable of being saprophytic

fallow cultivated land kept free from a crop or weeds during the normal growing season

fasciation malformation in shoots or floral organs manifested as enlargements and flattening such that several parts appear fused

fascicle (adj. fasciculate) small group, bundle, or cluster

fastidious in reference to prokaryotic organisms, having special growth and nutritional requirements

Federal Food, Drug, and Cosmetic Act (acronym FFDCA) the federal law that sets legal tolerances of pesticides on food products at harvest

Federal Insecticide, Fungicide, and Rodenticide Act (acronym FIFRA) the federal law that legislates the conditions for use of pesticides

feeder root fine root that absorbs water and dissolved nutrients

fermentation oxidation of certain organic substances in the absence of molecular oxygen

FFDCA (acronym for Federal Food, Drug, and Cosmetic Act) the federal law that sets legal tolerances of pesticides on food products at harvest

fiber elongated plant cell with thick walls (in xylem and phloem), dead at maturity

FIFRA (acronym for Federal Insecticide, Fungicide, and Rodenticide Act) the federal law that legislates the conditions for use of pesticides

filamentous (syn. filiform) threadlike

fission form of cell division in prokaryotes

fitness the ability of an organism to survive and reproduce; the ability of an organism to pass its genes to the next generation

flaccid wilted, lacking turgor

flag isolated, wilted, or necrotic branch with dead leaves attached

flagellum (pl. flagella) hairlike, whiplike, or tinsel-like appendage of a motile cell, bacterium, or zoospore that provides locomotion

fleck minute, discolored spot in green tissue

flexuous having turns or windings; capable of bending

flower break break or stripe in flower color (see also breaking)

focus (pl. foci) small area of diseased plants within a population

foliar pertaining to leaves

Food Quality Protection Act (acronym FQPA) the federal law that sets a health-based standard governing pesticide tolerance levels in food

forest decline combination of biotic factors (e.g., insect pests, pathogens) and abiotic factors (e.g., drought, air pollution) that contributes to poor growth and death of populations of trees

form genus a genus based on morphology and not on evolutionary relationships, such as used for imperfect fungi

forma specialis (abbrev. f. sp.; pl. *formae speciales*) a taxonomic group within a pathogenic species defined in terms of host range, i.e., members of different *formae speciales* infect different groups of plants

FQPA (acronym for Food Quality Protection Act) the federal law that sets a health-based standard governing pesticide tolerance levels in food

free water unbound water; often used to describe a film of water on a plant surface

fructification general term for a spore-bearing organ of a fungus

fruiting body any of various complex, spore-bearing fungal structures

fumigant (v. fumigate) a gas or volatile substance used to kill or inhibit the growth of microorganisms or other pests

Fungi Imperfecti archaic term for a group of fungi without a sexual stage; also, the asexual stage of ascomycetes and basidiomycetes (see also deuteromycetes, imperfect fungi)

fungicide (adj. fungicidal) chemical or physical agent that kills or inhibits the growth of fungi (contrasts with fungistat)

fungistasis inhibition of fungal growth, sporulation, or spore germination but not death; used to describe the nonspecific phenomenon in natural soils where spore germination is inhibited; often overcome by rhizosphere nutrients

fungistat (adj. fungistatic) a chemical or physical agent that inhibits fungal growth, sporulation, or spore germination, but does not cause death (contrasts with fungicide)

fungus (pl. fungi) a eukaryotic organism that is usually filamentous (forming a mycelium) and heterotrophic, has cell walls composed of chitin, and reproduces by sexual and/or asexual spores

fusiform spindle-shaped; tapering at each end

gall abnormal swelling or localized outgrowth, often roughly spherical, produced by a plant as a result of attack by a fungus, bacterium, nematode, insect, or other organism (see also tumor)

gallery insect tunnel in bark and wood

gametangium (pl. gametangia) cell containing gametes or nuclei that act as gametes

gamete sex cell

gametophyte the haploid stage of a plant (contrasts with sporophyte)

gene unit within an organism controlling heritable characteristics; genes are organized on chromosomes

gene gun a device used to bombard plant cells with metal particles coated with foreign genes to accomplish genetic engineering (see also biolistic transformation)

gene-for-gene hypothesis the hypothesis that corresponding genes for resistance and virulence exist in the host and pathogen, respectively

gene silencing the switching off of a gene by a process other than genetic modification; one example is the disabling of a gene by small interfering RNAs (siRNAs) that target messenger RNA for destruction

general resistance resistance that is effective against all biotypes of the pathogen (see also durable resistance, horizontal resistance, race-nonspecific resistance) (contrasts with specific resistance, vertical resistance)

genetic relating to heredity; referring to heritable characteristics

genetic code the system of triplet codons composed of nucleotides of DNA or RNA that determine the amino acid sequence of a protein

genetic engineering the transfer of specific genes between organisms using enzymes and laboratory techniques rather than biological hybridization

genetically modified organism (acronym GMO) organism possessing a gene from another species; used to describe the organisms that have been the subject of genetic engineering (see also transgenic)

genome the complete genetic information of an organism or virus

genotype genetic constitution of an individual or group; class or group of individuals sharing a specific genetic makeup (contrasts with phenotype)

genus (pl. genera) a taxonomic category that includes a group of closely related (structurally or phylogenetically) species; the genus or generic name is the first name in a Latin binomial

geocarp fruit that ripens beneath the ground (e.g., peanut pod)

germ theory the theory that infectious or contagious diseases are caused by microorganisms (germs)

germ tube hypha resulting from an outgrowth of the spore wall and cytoplasm after germination

germinate (n. germination) begin growth (as of a seed, spore, sclerotium, or other reproductive body)

germplasm bearer of heredity material, often loosely applied to cultivars and breeding lines

giant cell enlarged, multinucleate cell formed in roots by repeated nuclear division without cell division; induced by secretions of certain sedentary plant-parasitic nematodes

gibberellin plant hormone (growth regulator) that affects stem elongation

gill thin, radial membrane producing basidiospores in the cap of certain basidiomycetous fungi (e.g., mushrooms)

girdle to circle and cut through a stem or the bark and outer few rings of wood, disrupting the phloem and xylem

glabrous smooth; without hairs

glucoside a substance that, on decomposition, yields glucose and certain other compounds; some glucosides are defense compounds produced by plants, e.g., cyanogenic or phenolic glucosides

GMO (acronym for genetically modified organism) organism possessing a gene from another species; used to describe the organisms that have been the subject of genetic engineering (see also transgenic)

gonad sexual structure in animals that produces gametes

graft transfer of aerial parts of one plant (e.g., buds or twigs - the scion) into close cambial contact with the root or trunk (the rootstock) of a different plant; a method of plant propagation; the joining of cut surfaces or growing roots of two plants to form a living union

graft transmission transmission of a pathogen from one host plant to another through fusion of living tissue from the diseased host with living tissue of a healthy host

gram-negative pertaining to bacteria staining red or pink after treatment with Gram's stain

gram-positive pertaining to bacteria staining violet or purple after treatment with Gram's stain

Gram stain procedure used for identification of bacteria in which crystal violet stain, Gram's iodine, ethyl alcohol, and safranin stain are applied in succession to cells of the bacteria

gravid containing an egg or eggs; capable of depositing eggs

green manure a crop plowed under while still green and growing to improve the soil; sometimes used to enhance populations of antagonistic microorganisms for biological control

growth regulator a chemical substance produced in one part of an organism and transported in minute quantities to induce a growth response in another part, e.g., in plants, auxins, cytokinins, and gibberellins (see also hormone)

guard cells paired, specialized, epidermal cells that contain chloroplasts and surround a stoma

gum gelatinous, sugary aggregate that is synthesized and exuded by plant tissues

gummosis (pl. gummoses) pathologic condition characterized by excessive formation of gums; the products of cell degeneration

guttation exudation of watery, sticky liquid from hydathodes, especially along leaf margins

habitat a place with a particular kind of environment suitable for the growth of an organism

haploid having a single complete set of chromosomes (contrasts with dikaryotic, diploid, polyploid)

hardiness ability to withstand stress

haulm stems or stalks, collectively

haustorium (pl. haustoria) specialized branch of a parasite formed inside host cells to absorb nutrients

head in plants, a type of inflorescence in which numerous small flowers are densely crowded on a disk or around a central stalk

heartwood central cylinder of nonfunctional xylem in a woody stem (contrasts with sapwood)

hemiparasite parasitic flowering plant that contains chlorophyll when mature (photosynthetic) and obtains water with dissolved nutrients by connecting to the host xylem via the haustorium, e.g., true mistletoe (*Phoradendron* spp. or *Viscum* spp.) or witchweed (*Striga* spp.) (contrasts with holoparasite)

herbaceous describing primary, soft, nonwoody tissue, as a plant or plant part; having the characteristics of an herb

herbicide a chemical used for killing plants or inhibiting plant growth, e.g., a weed or grass killer

heritability a measure of the ability of a characteristic to be passed from one generation to the next

hermaphrodite (adj. hermaphroditic) an organism having both male and female reproductive organs

heteroecious pertaining to a rust fungus requiring two unrelated host plants for completion of its life cycle (contrasts with autoecious)

heterokaryon (adj. heterokaryotic) cell with genetically different nuclei

heterosis increased vigor of hybrid offspring when compared to either parent (see also hybrid vigor)

heterothallism (adj. heterothallic) condition in which sexual reproduction can occur only in the presence of genetically different mycelia (contrasts with homothallism)

heterotroph an organism that obtains nourishment from outside sources and must obtain its carbon from organic carbon compounds (contrasts with autotroph)

heterozygous having alternate forms (alleles) of a gene on homologous chromosomes (contrasts with homozygous)

holomorph the whole fungus in all its states (contrasts with anamorph, imperfect state, perfect state, teleomorph)

holoparasite parasitic flowering plant that lacks chlorophyll (nonphotosynthetic) and must rely totally on the contents of the xylem and the phloem of the host plant, e.g., broomrape (*Orobanche* spp.) (contrasts with hemiparasite)

homothallism (adj. homothallic) condition in which sexual reproduction occurs with a single thallus; self-fertile (contrasts with heterothallism)

homozygous having the same form (allele) of a gene on homologous chromosomes (contrasts with heterozygous)

honeydew sugary ooze or exudate, often from aphids, and a characteristic symptom of ergot

horizontal resistance resistance that is effective against all biotypes of the pathogen (see also durable resistance, general resistance, race-nonspecific resistance) (contrasts with specific resistance, vertical resistance)

hormone a chemical substance produced in one part of an organism and transported in minute quantities to induce a growth response in another part, e.g., in plants, auxins, cytokinins, and gibberellins (see also growth regulator)

host plant living plant attacked by or harboring a parasite or pathogen and from which the invader obtains part or all of its nourishment

host range the range of plants on which an organism, particularly a parasite, feeds

HR (acronym for hypersensitive response) rapid and localized cell death at the site of infection in resistant interactions between plants and pathogens

hyaline transparent or nearly so; translucent; often used in the sense of colorless

hybrid (v. hybridize) offspring of two individuals of different genotypes

hybrid vigor increased vigor of hybrid offspring when compared to either parent (see also heterosis)

hybridoma a cell produced by the fusion of an antibody-producing cell and a lymphoma (cancer) cell for production of monoclonal antibodies

hydathode epidermal leaf structure specialized for secretion or exudation of water; leaf opening at the terminus of a vein

hydrogen fluoride (HF) a common pollutant from volcanoes and several industries such as brick factories, aluminum smelters, and phosphate fertilizer factories

hymenium continuous, spore-bearing layer of a fungus fruiting body

hyperparasite organism that is parasitic upon another parasite

hyperplasia (adj. hyperplastic) abnormal increase in the number of cells, often resulting in the formation of galls or tumors

hypersensitive extremely or excessively sensitive; often refers to an extreme reaction to a pathogen

hypersensitive response (acronym HR) rapid and localized cell death at the site of infection in resistant interactions between plants and pathogens

hypersensitive response and pathogenicity (*hrp*) gene gene in a bacterial pathogen required for elicitation of the hypersensitive (HR) response in resistant plants and causation of disease in susceptible plants; encodes a protein in the type III secretion system

hypertrophy (adj. hypertrophic) abnormal increase in the size of cells in a tissue or organ, often resulting in the formation of galls or tumors

hypha (pl. hyphae; adj. hyphal) single, tubular filament of a fungal thallus or mycelium; the basic structural unit of a fungus

hyphal sheath dense hyphal mass of an ectomycorrhizal fungus enclosing the short feeder roots of plants (see also mantle)

hyphomycetes archaic term for imperfect fungi that produce conidia freely or on a sporodochium or synnema (contrasts with coelomycetes)

hyphopodium stalked, thick-walled, lobed cells that stick to plant surfaces; sometimes used to describe the infection structures produced by ectotrophic hyphae of certain root-infecting fungi such as *Gaeumannomyces* (take-all pathogen)

hypocotyl portion of the stem below the cotyledons and above the root (contrasts with epicotyl)

hypovirulence reduced ability to cause disease

icosahedral (n. icosahedron) having 20 faces, as a polyhedral virus particle (see also isometric)

immune cannot be infected by a given pathogen

immunoassay a detection method based on antibodies specifically selected to react with the substance to be detected (the antigen)

immunosupressant chemical or activity that suppresses the natural immune responses in animals, including humans

imperfect fungi archaic term for a group of fungi without a sexual stage; also, the asexual stage of ascomycetes and basidiomycetes (see also deuteromycetes, Fungi Imperfecti)

imperfect state the asexual form in the life cycle of a fungus, when asexual spores (such as conidia) or no spores are produced (see also anamorph) (contrasts with holomorph, perfect state, teleomorph)

in planta in a plant

in situ in its original place or environment

in vitro in glass, on artificial media, or in an artificial environment; outside the host

in vivo within a living organism

inclusion body structure developed within a plant cell as a result of infection by a virus, often useful in identifying the virus

incomplete dominance the interaction of alleles of a gene that produces an intermediate phenotype, as in the production of pink flowers when red and white alleles are present in a heterozygous individual

incubation period the time between penetration of a host by a pathogen and the first appearance of disease symptoms; the time during which microorganisms inoculated onto a medium are allowed to grow

indehiscent not opening or with no special method of opening, as in anthers, asci, fruiting bodies, fruits, sporangia, etc. (contrasts with dehiscent)

indeterminate continuing to grow vegetatively while producing flowers or reproductive structures (contrasts with determinate)

indexing testing of a plant for infection, often by mechanical transmission or by grafting tissue from it to an indicator plant

indicator plant plant that reacts to a pathogen or an environmental factor with specific symptoms and is used to detect or identify the pathogen or determine the effects of the environmental factor

indirect penetration penetration of plant tissues by a pathogen through natural openings (e.g., stomata) or wounds (contrasts with direct penetration)

induced produced in response to a stimulus (contrasts with constitutive)

induced systemic resistance (acronym ISR) reduced disease symptoms on a portion of a plant distant from the area where the inducing agent is active, caused by the triggering of active plant defenses against a variety of pathogens; used to describe increased resistance in plants induced by certain rhizobacteria (see also systemic acquired resistance)

infect (n. infection) to enter, invade, or penetrate and establish a parasitic relationship with a host plant

infection court site in or on a host plant where infection can occur

infection cushion organized mass of hyphae formed on the surface of a plant from which numerous infective hyphae develop

infection focus initial site of infection, followed by secondary spread, generally with reference to a population of plants

infection peg the specialized, narrow hyphal strand on the underside of an appressorium that penetrates host cells (see also penetration peg)

infection period the time required for infection to occur under conducive environmental conditions, usually hours of leaf wetness and temperature

infectious pertaining to a disease capable of spreading from plant to plant (see also biotic) (contrasts with abiotic, noninfectious)

infective referring to an organism able to attack a host and cause infection; referring to a vector carrying or containing a pathogen and able to transfer it to a host plant

infest (n. infestation) to attack as a pest (used especially of insects and nematodes); to contaminate, as with microorganisms; to be present in large numbers

inflorescence flower or flower cluster

initial inoculum inoculum, usually from an overwintering source, that initiates disease in the field, as opposed to inoculum that spreads disease during the season (see also primary inoculum) (contrasts with secondary inoculum)

injury damage caused by transitory interaction with an agent such as an insect, chemical, or unfavorable environmental condition

inner bark interior, living portion of the bark; secondary phloem (contrasts with outer bark)

inoculate (n. inoculation) to place inoculum in an infection court; to insert a pathogen into healthy tissue

inoculum (pl. inocula) pathogen or its parts, capable of causing infection when transferred to a favorable location

inoculum density a measure of the number of propagules of a pathogenic organism per unit area or volume

insect member of the class Hexapoda (phylum Arthropoda) possessing three sets of limbs attached to a central body segment

instar one of the stages of growth between molts, in the life history of certain insects

integrated pest management (acronym IPM) a combination of strategies to reduce losses caused by pests and pathogens based on environmental and economic considerations

intercellular between or among cells

intercrop to grow two or more crops simultaneously on the same area of land

internode (adj. internodal) the portion of a stem between two successive nodes

internal transcribed spacers (acronym ITS) regions of a DNA sequence that separate genes for certain ribosomal RNAs (rRNAs). These genes appear to be nearly identical among a wide variety of species, but the DNA spacers between them are quite variable and may be species specific. This makes them good potential targets for “primers” for polymerase chain reaction (PCR) amplification, which is used to identify a wide variety of organisms.

interveinal between (leaf) veins

intracellular through or within cells

intumescence blisters produced on leaves and other plant parts under conditions of high moisture and restricted transpiration (see also edema)

IPM (acronym for integrated pest management) a combination of strategies to reduce losses caused by pests and pathogens based on environmental and economic considerations

isolate (n.) a culture or subpopulation of a microorganism separated from its parent population and maintained in some sort of controlled circumstance; (v.) to remove from soil or host material and grow in pure culture

isometric usually used to describe virus particles that are icosahedral in structure and appear approximately round (see also icosahedral)

ITS (acronym for internal transcribed spacer) region of a DNA sequence that separates genes for certain ribosomal RNAs (rRNAs). These genes appear to be nearly identical among a wide variety of species, but the DNA spacers between them are quite variable and may be species specific. This makes ITS regions good potential targets for “primers” for polymerase chain reaction (PCR) amplification, which is used to identify a wide variety of organisms.

juvenile an immature form that appears similar to but usually smaller than the adult and is not sexually mature (e.g., insects with gradual metamorphosis, nematodes)

karyogamy the fusion of nuclei

Koch’s postulates the procedure used to prove the pathogenicity of an organism, i.e., its role as the causal agent of a disease

knot gall; localized abnormal swelling

lamina expanded part of a leaf (contrasts with petiole)

land race plant stock selected by farmers on a local basis over many years

larva (pl. larvae) immature stage of certain animals (especially insects) that undergo complete metamorphosis; the wormlike or caterpillar stage of the life cycle of such insects

latent present but not manifested or visible, as a symptomless infection by a pathogen

latent infection infection unaccompanied by visible symptoms

latent period the time between infection and the production of new inoculum; the time after a vector has acquired a pathogen and before it can be transmitted

lateral bud bud that develops in the axil of a leaf (see also axillary bud)

LD_{50} (abbrev. for lethal dose 50%) a measure of relative acute toxicity; the dose of a compound that causes death in 50% of the test animals treated

leaf dip inspection of a sample of sap from a suspected virus-infected leaf using an electron microscope

leaf sheath lower, tubular part of a grass leaf that clasps the culm

leaf spot a plant disease lesion typically restricted in development in the leaf after reaching a characteristic size

leafhopper mobile insect with sucking mouthparts (order Hemiptera)

leaflet one of the separate blades or divisions of a compound leaf

leafroll disease symptom in which the edges of the leaf roll or turn, either up or down; often a symptom of virus infection

legume simple, dry, dehiscent fruit developed from a simple pistil and splitting at maturity along two seams; a plant of the family Fabaceae (formerly Leguminosae)

lenticel a natural opening in the surface of a stem or tuber, fruit or root for gas exchange

lesion localized diseased area or wound

life cycle the cyclical stages in the growth and development of an organism

lignification hardening of tissue through the deposition of lignin in the cell wall

lignin complex organic substance or group of substances that impregnate the cell walls of xylem vessels and certain other plant cells; major constituent of wood

local lesion small, restricted lesion, often the characteristic reaction of differential cultivars to specific pathogens, especially in response to mechanical inoculation with a virus

lodge to fall over

lumen (pl. lumina) central cavity of a cell or other structure

macerate to cause disintegration of tissues by separation of cells; to soften by soaking

macroconidium (pl. macroconidia) the larger of two kinds of conidia formed by certain fungi (contrasts with microconidium)

macrocyclic pertaining to a rust fungus that typically exhibits all five stages of the rust life cycle (contrasts with demicyclic, microcyclic)

macronutrient an element needed in relatively large quantities for plant growth, e.g., nitrogen (N), phosphorus (P), and potassium (K) (contrasts with micronutrient)

MAMPS (acronym for microbe-associated molecular patterns) molecular signals or elicitors from microbes, including pathogens, that are recognized by plant or animal receptors and can influence innate immunity of the host (see also pathogen-associated molecular patterns, acronym PAMPS)

mantle dense hyphal mass of an ectomycorrhizal fungus enclosing the short feeder roots of plants (see also hyphal sheath)

marker-assisted selection a procedure used in plant breeding in which genetic markers that are (a) easy to identify and (b) linked to desirable genetic traits that are difficult to identify (such as disease resistance) are used to aid in selection from a population

maternal inheritance inheritance of nonnuclear genes, i.e., those in mitochondria and chloroplasts (see also cytoplasmic inheritance, extrachromosomal inheritance)

mating types compatible strains, usually designated + and – or A and B, necessary for sexual reproduction in heterothallic fungi

mechanical injury injury of a plant part by abrasion, mutilation, or wounding

mechanical transmission spread or introduction of inoculum to an infection court (wounding) by human manipulation, accompanied by physical disruption of host tissues

medium (pl. media) a mixture of organic and/or inorganic chemical compounds and water that provides the nutrients needed for the growth of a microorganism in vitro; for higher plants, a mixture of fertilizers and other components in which a plant is growing

meiosis process of nuclear division in which the number of chromosomes per nucleus is halved, i.e., converting the diploid state to the haploid state (contrasts with mitosis)

melanin brown-black pigment; common in sclerotia and other survival structures

meristem (adj. meristematic) plant tissue characterized by frequent cell division, producing cells that become differentiated into specialized tissues

meristem culture aseptic in vitro culture of a plant or plant part from a portion of the meristem; a method used to produce pathogen-free plants

messenger RNA (abbrev. mRNA) a form of RNA that carries information to direct the synthesis of protein

metabolite any chemical participating in or resulting from metabolism; a nutrient

microbe an organism of such small size that it can be seen only as an individual organism with the aid of a microscope (see also microorganism)

microbe-associated molecular patterns(acronym MAMPS) molecular signals or elicitors from microbes, including pathogens, that are recognized by plant or animal receptors and can influence innate immunity of the host (see also pathogen-associated molecular patterns, acronym PAMPS)

microbial pertaining to or relating to microbes or microorganisms

microclimate weather conditions on a small scale, e.g., at the surface of the plant or within a crop

microconidium (pl. microconidia) the smaller of two kinds of conidia formed by certain fungi (contrasts with macroconidium)

microcyclic describing a rust fungus that produces only teliospores and basidiospores (contrasts with demicyclic, macrocyclic)

microflora the combination of all microorganisms in a particular environment

micron (syn. micrometer) one millionth of a meter

micronutrient an essential element needed by plants for growth and required in very small amounts (contrasts with macronutrient)

microorganism an organism of such small size that it can be seen only as an individual organism with the aid of a microscope (see also microbe)

microsclerotium (pl. microsclerotia) microscopic, dense aggregate of darkly pigmented, thick-walled hyphal cells

middle lamella the layer, consisting largely of pectic substances, between the walls of adjacent plant cells

midrib central, thickened vein of a leaf

migratory migrating from place to place on a plant or from plant to plant when feeding (contrasts with sedentary)

mildew thin coating of mycelial growth and spores on the surfaces of infected plant parts

mitochondrion (pl. mitochondria) cellular organelle outside the nucleus that functions in respiration

mitosis nuclear division in which the chromosome number remains the same (contrasts with meiosis)

MLO (acronym for mycoplasmalike organism) archaic term for phytoplasma; plant-parasitic pleomorphic mollicute (prokaryote with no cell wall) found in phloem tissue; cannot yet be grown on artificial nutrient media (see also phytoplasma)

mold any microfungus with conspicuous, profuse, or woolly superficial growth (mycelium and/or spore masses) on various substrates, especially an economically important saprobe; molds commonly grow on damp or decaying matter and on the surfaces of plant tissues

mollicute one of a group of prokaryotic organisms bounded by flexuous membranes and lacking cell walls (see also phytoplasma, spiroplasma)

molt to shed a cuticle or body encasement during a phase of growth

monoclonal antibody antibody produced from clones of a single antibody-producing cell (contrasts with polyclonal antibody)

monocot (syn. monocotyledon) plant with one cotyledon, such as grasses, grain crops, corn (contrasts with dicot)

monoculture the growth of the same plant species in close proximity, with few or no other types of plant present (contrasts with polyculture)

monocyclic having one disease or life cycle per growing season (contrasts with polycyclic)

monoecious having male and female organs on the same individual; in plants, often refers to separate male and female flowers on the same individual (contrasts with dioecious)

monogenic determined by a single gene (contrasts with polygenic)

monogenic resistance resistance conferred by a single gene (see also single gene resistance) (contrasts with multigenic resistance, oligogenic resistance, polygenic resistance)

monoxenic culture a culture containing one species of organism growing in the presence of one other species of organism

morphology (adj. morphologic) the study of the form of organisms; form and structure of organisms

mosaic disease symptom characterized by nonuniform coloration, with intermingled normal, light green and yellowish patches, usually caused by a virus (see also mottle)

motile capable of self-propulsion by means of flagella, cilia, or amoeboid movement

mottle disease symptom comprising light and dark areas in an irregular pattern, usually caused by a virus (see also mosaic)

movement protein for viruses, a protein encoded by the pathogen that is required for spread within an infected plant

mRNA (abbrev. for messenger RNA) a form of RNA that carries information to direct the synthesis of protein

mucilaginous viscous, slimy

mulch layer of material, such as organic matter or plastic, applied to the surface of the soil for purposes such as retention of water and inhibition of weeds

multicellular composed of many cells (contrasts with unicellular)

multigenic resistance resistance conferred by several genes (see also polygenic resistance) (contrasts with monogenic resistance, oligogenic resistance, single gene resistance)

multiline mixture of cultivars that differ only in single, major genes for resistance to a pathogen, giving the opportunity to modify the mixture from year to year as needed

multinucleate having more than one nucleus per cell (contrasts with uninucleate)

multiparticulate virus plant virus in which the genome is divided into more than one piece of nucleic acid encapsidated in more than one particle

multipartite virus plant virus in which the genome is divided into more than one piece of nucleic acid

multiseptate having many septa or crosswalls

mummy a dried, shriveled fruit; plant part or organ partially or completely replaced with fungal structures

mushroom a fleshy fruiting body of a fungus, especially of a basidiomycete of the family Agaricaceae

mutagen a substance that causes a mutation

mutation (n. mutant) an abrupt heritable or genetic change in a gene or an individual as a result of an alteration in a gene or chromosome, or of an increase in chromosome number

Mycelia Sterilia archaic term for a group in the Fungi Imperfecti (also an archaic term) that does not produce spores, but may produce chlamydospores or sclerotia, e.g., *Rhizoctonia* and *Sclerotium*; subsequent research has defined sexual reproduction for some of these species

mycelium (pl. mycelia, adj. mycelial) mass of hyphae constituting the body (thallus) of a fungus

mycology the study of fungi

mycoparasite a fungus that attacks another fungus

mycoplasmalike organism (acronym MLO) archaic term for phytoplasma; plant-parasitic pleomorphic mollicute (prokaryote with no cell wall) found in phloem tissue; cannot yet be grown on artificial nutrient media (see also phytoplasma)

mycorrhiza (pl. mycorrhizae; adj. mycorrhizal) symbiotic association between a nonpathogenic or weakly pathogenic fungus and the roots of plants (see also ectomycorrhiza, endomycorrhiza)

mycotoxin poisonous compound produced by a fungus

mycovirus a virus that infects fungi

myxomycetes a group of nonpathogenic fungus-like organisms that form a vegetative amoeboid plasmodium and spores (see also slime molds)

nanometer one billionth of a meter; used in measurement of viruses

necrosis (adj. necrotic) death of cells or tissue, usually accompanied by darkening to black or brown

necrotroph a parasite that typically kills and obtains its energy from dead host cells (contrasts with biotroph, obligate parasite)

nectary a nectar-secreting gland in a flower

needlecast (of conifers) disease symptom caused by fungi that results in premature drop of needles

negative-sense RNA (–RNA) ribonucleic acid complementary to the positive or plus sense; not translated into protein (contrasts with positive-sense RNA)

nematicide agent, usually a chemical, that kills nematodes

nematode nonsegmented roundworm (animal), parasitic on plants or animals, or free living in soil or water

nitrogen cycle the cycling of the element nitrogen from gaseous forms to various inorganic forms, such as ammonium, nitrates, and nitrites, and organic forms, such as nucleic acids and proteins, and back to elemental nitrogen

nitrogen fixation the conversion of gaseous N_2 to a form available for plant uptake

nitrogen oxide (NO_x) primary air pollutant produced from internal combustion engines and other industrial processes

node (adj. nodal) enlarged portion of a shoot at which leaves or buds arise

nodule (v. nodulation) small knot or irregular, rounded lump; on leguminous plants, a structure on a root that contains nitrogen-fixing bacteria

nomenclature the scheme by which names are attached to objects, including organisms

noninfectious pertaining to a disease that is caused by an abiotic agent that cannot be transmitted from one plant to another (see also abiotic) (contrasts with biotic, infectious)

nonpathogenic unable to cause disease (see also avirulent)

nonpersistent transmission a type of virus transmission in which the virus is acquired and transmitted by the vector after short feeding times and is retained by the vector for only a short period of time (see also stylet-borne transmission) (contrasts with circulative transmission, persistent transmission, propagative transmission)

nonseptate without cross walls (see also aseptate, coenocytic)

no-till a cultural system most often used with annual crops, in which the new crop is seeded or planted directly in a field on which the preceding crop plants were cut down, had the tops harvested, or were destroyed by a nonselective herbicide

nucleic acid genetic material of all living organisms, including DNA (deoxyribonucleic acid) and RNA (ribonucleic acid)

nucleoid region in prokaryotes, chloroplasts, and mitochondria where the DNA is concentrated. Unlike a nucleus, it is not bound by a membrane.

nucleus dense aggregation of proteinaceous matter and nucleic acid in cells, surrounded by a membrane; contains chromosomes and controls heredity

nucleotide a subunit of a nucleic acid, either DNA or RNA

obligate parasite organism that can grow only as a parasite in association with its host plant and cannot be grown in artificial culture media (see also biotroph) (contrasts with necrotroph)

occlusion block or plug that stops flow of liquids (as in vessels)

oedema (also edema) blisters produced on leaves and other plant parts under conditions of high moisture and restricted transpiration (see also intumescence)

oligogenic resistance resistance conferred by a few genes (contrasts with monogenic resistance, multigenic resistance, polygenic resistance, single gene resistance)

oogonium (pl. oogonia) female gametangium of oomycetes, containing one or more gametes

oomycetes (adj. oomycetous) group of fungus-like organisms typically with nonseptate mycelium, asexual sporangia and zoospores, and sexual oospores, now comprising the phylum Oomycota in the kingdom Straminipila

oospore thick-walled, sexually derived resting spore of oomycetes

ooze mass of bacterial cells mixed with host fluids

open reading frame portion of a nucleic acid molecule that is translated into a protein

organelle a membrane-bound structure within a cell having a specialized function, e.g., a mitochondrion or chloroplast

organic describing a molecule containing carbon atoms; pertaining to living organisms

ostiole (adj. ostiolate) pore; opening in the papilla or neck of a perithecium, pseudothecium, or pycnidium through which spores are released

outer bark exterior, nonliving portion of bark in woody plants (contrasts with inner bark)

ovary the female reproductive structure of organisms; in plants, enlarged basal portion of a pistil, containing the ovules and developing into the fruit

overseason to survive or persist from one planting season to the next

overwinter to survive or persist through the winter period

oviposit to deposit or lay eggs with an ovipositor

ovule enclosed structure that, after fertilization, becomes a seed; an egg contained within an ovary

ozone (O_3) a secondary air pollutant that is a highly reactive form of oxygen formed when the exhaust of automobiles and other internal combustion engines is in the presence of sunlight

ozone layer a protective layer of ozone in the upper atmosphere that reduces ultraviolet radiation

palisade parenchyma tissue found beneath the upper epidermis of leaves, composed of elongate, tubular cells arranged upright, in the manner of posts in a palisade fortification

PAMPS (acronym for pathogen-associated molecular patterns) molecular signals or elicitors from pathogens that are recognized by plant or animal receptors and can influence innate immunity of the host (see also microbe-associated molecular patterns, acronym MAMPS)

PAN (acronym for peroxyacetyl nitrate) a secondary air pollutant formed when the exhaust of automobiles and other internal combustion engines is in the presence of sunlight

pandemic a widespread and destructive outbreak of disease occurring simultaneously in several countries

papilla (pl. papillae, adj. papillate) nipplelike projection; a structure formed at the tip of some sporangia; also, the localized wall thickenings on the inner surface of plant cell walls at sites penetrated by fungi

paragynous having the antheridium contact the oogonium on the side, as in many *Pythium* spp. (contrasts with amphigynous)

parasexualism a type of recombination, found in certain heterokaryotic fungi, that is based on mitosis rather than meiosis. Genetically distinct haploid nuclei fuse in the heterokaryon. The resulting diploid nuclei multiply by mitotic division, with some crossing over, and a diploid homokaryon develops. During repeated mitotic divisions, chromosomes may be shed by the diploid nucleus to produce a haploid nucleus.

parasite (adj. parasitic) organism that lives in intimate association with another organism on which it depends for its nutrition; not necessarily a pathogen

parenchyma (adj. parenchymatous) soft tissue of living plant cells with undifferentiated, thin, cellulose walls

parthenogenesis (adj. parthenogenetic; sometimes parthenogenic) reproduction by the development of an unfertilized egg

partial resistance resistance in which disease develops more slowly, or to a lesser extent, on the host, but the host does become diseased (see complete resistance)

pasteurization process used to free a material, usually a liquid, of selected harmful microorganisms using heat

pathogen (adj. pathogenic) a disease-producing organism or agent

pathogen-associated molecular patterns (acronym PAMPS) molecular signals or elicitors from pathogens that are recognized by plant or animal receptors and can influence innate immunity of the host (see also microbe-associated molecular patterns, acronym MAMPS)

pathogenesis production and development of disease

pathogenesis-related (PR) proteins proteins, such as antimicrobial proteins and hydrolytic enzymes, that are synthesized in the early events of the plant defense response

pathogenicity ability to cause disease

pathology the study of diseases

pathotype a subdivision of a pathogen species characterized by its pattern of virulence or avirulence to a series of differential host varieties

pathovar (abbrev. pv.) a subdivision of a plant-pathogenic bacterial species defined by host range; pathovar for bacteria is equivalent to *forma specialis* for fungi

PCR (acronym for polymerase chain reaction) a technique used to amplify the number of copies of a specific region of DNA in order to produce enough of the DNA for use in various applications, such as identification and cloning

pectin a methylated polymer of galacturonic acid found in the middle lamella and the primary cell wall of plants; jelly-forming substance found in fruit

pedicel small slender stalk; stalk bearing an individual flower, inflorescence, or spore

peduncle stalk or main stem of an inflorescence; part of an inflorescence or a fructification

penetration initial invasion of a host by a pathogen

penetration peg the specialized, narrow hyphal strand on the underside of an appressorium that penetrates host cells (see also infection peg)

penicillin antibiotic produced by certain species of *Penicillium* and effective against gram-positive bacteria

perennial something that occurs year after year; a plant that survives for several to many years (contrasts with annual, biennial)

perfect flower flower possessing both stamens and pistils

perfect state the sexual form in the life cycle of a fungus (see also teleomorph) (contrasts with anamorph, holomorph, imperfect state)

pericarp fruit wall

pericycle layer or layers of cells between the phloem and the endodermis of roots, giving rise to branch roots

perithecium (pl. perithecia) flask-shaped or subglobose, thin-walled fungus fruiting body (ascocarp) containing asci and ascospores; spores are expelled or released through a pore (ostiole) at the apex

peritrichous having hairs or flagella distributed over the whole surface (contrasts with polar)

peroxyacetyl nitrate (acronym PAN) a secondary air pollutant formed when the exhaust of automobiles and other internal combustion engines is in the presence of sunlight

persistent transmission a type of virus transmission in which the virus is acquired and transmitted by the vector after relatively long feeding times and remains transmissible for a prolonged period while in association with its vector (see also circulative transmission, propagative transmission) (contrasts with nonpersistent transmission, stylet-borne transmission)

pest any organism that damages plants or plant products

pesticide a chemical used to kill pests

petiole stalk portion of a leaf (contrasts with lamina)

pH negative logarithm of the effective hydrogen ion concentration; a measure of acidity (pH 7 is neutral; values less than pH 7, acidic; greater than pH 7, alkaline or basic)

phellem cork; a protective tissue composed of nonliving cells with suberized walls, produced by the phellogen and accumulating on the surface of stems and roots

phelloderm tissue formed by and internal to the phellogen; resembles the cortical parenchyma in morphology

phellogen cork cambium; lateral meristem forming the periderm, a protective tissue in stems and roots; phellem (cork) is produced toward the surface, phelloderm toward the inside

phenological synchrony the coordinated development of parasites and their hosts based on degree-days and other environmental factors such that parasites can successfully infect their hosts; e.g., the production and release of ascospores of *Venturia inaequalis*, the apple scab pathogen, from fallen leaves from the previous season, when new apple leaves are expanding

phenotype external, visible physical characteristics of an organism determined by the interaction of its genotype with the environment (contrasts with genotype)

pheromone chemical substance that attracts members of the same species or one sex of that species, esp. insects and nematodes

phialide end cell of a conidiophore with one or more open ends through which a basipetal succession of conidia develops

phloem food-conducting, food-storing tissue in the vascular system of roots, stems, and leaves

phloem necrosis death of phloem cells, often visible, caused by infection by systemic phloem pathogens such as phytoplasmas, spiroplasmas, and viruses

photochemical reaction chemical reaction that requires sunlight

photosynthate product of photosynthesis

photosynthesis manufacture of carbohydrates from carbon dioxide and water in the presence of chlorophyll(s), using light energy and releasing oxygen

phycomycete archaic term for a member of a group of fungi without cross walls (septa) in their mycelia

phyllody change of floral organs to leaflike structures

phylloplane leaf surface

phylloplane-competent pertaining to epiphytic microorganisms adapted to living on the leaf surface

phyllotaxy arrangement of leaves on a stem in relation to one another

phytoalexin substance produced in higher plants in response to a number of chemical, physical, and biological stimuli that inhibits the growth of certain microorganisms

phytopathogenic able to cause disease in plants

phytopathology the study of plant diseases (see also plant pathology)

phytoplasma plant-parasitic pleomorphic mollicute (prokaryote with no cell wall) found in phloem tissue; cannot yet be grown on artificial nutrient media (see also mycoplasmalike organism, acronym MLO)

phytosanitary certificate official document that indicates that plant material has been inspected and found to be free of certain pathogens

phytotoxic harmful to plants (usually used to describe chemicals)

pigment a colored compound, such as chlorophyll, in the cells of plants or fungi

pistil the ovule-bearing organ of the plant consisting of the ovary and its appendages (e.g., style, stigma)

pith parenchymatous tissue occupying the center of the stem

plant pathology the study of plant diseases (see also phytopathology)

plasmid a circular, self-replicating hereditary element that is not part of a chromosome; plasmids are used in recombinant DNA experiments as acceptors and vectors of foreign DNA

plasmodesma (pl. plasmodesmata) a cytoplasmic strand that connects living cells

plasmodiophoromycetes a group of fungus-like organisms, sometimes called the Plasmodiophoromycota or the endoparasitic slime molds, characterized by the production of zoospores and plasmodia that are restricted to the cells of their host

plasmodium (pl. plasmodia) naked, multinucleate mass of protoplasm moving and feeding in amoeboid fashion

plasmogamy the fusion of two sex cells

plasmolysis shrinking of a protoplast resulting from water loss from the vacuole of a plant cell

pleomorphic able to assume various shapes (and perhaps sizes); pertaining to a life cycle in which an organism has more than one distinct form

ploidy the number of (complete) sets of chromosomes in a cell, e.g., haploid, diploid, polyploid

polar at one end or pole of the cell, as for flagella (contrasts with peritrichous)

pollen male sex cells produced by anthers of flowering plants or within the male cones of seed plants that produce them

pollination transfer of pollen from anther to stigma or from a staminate cone to an ovulate cone

polyclonal antibody a preparation containing antibodies against more than one epitope of an antigen (contrasts with monoclonal antibody)

polyculture production of multiple crops in the same space, in imitation of the diversity of natural ecosystems (contrasts with monoculture)

polycyclic having several to many disease cycles in a growing season (contrasts with monocyclic)

polyetic requiring more than one year to complete one life or disease cycle

polygenic pertaining to, or governed by, many genes (contrasts with monogenic)

polygenic resistance resistance conferred by several genes (see also multigenic resistance) (contrasts with monogenic resistance, oligogenic resistance, single gene resistance)

polymerase chain reaction (acronym PCR) a technique used to amplify the number of copies of a specific region of DNA in order to produce enough of the DNA for use in various applications such as identification and cloning

polyploid having three or more complete sets of chromosomes (contrasts with haploid, dikaryotic, diploid)

polyprotein a protein translated from an entire viral genome, which is then cleaved by proteases (protein-degrading enzymes) into the active protein products

pome (syn. pome fruit) simple, fleshy, indehiscent fruit derived from several carpels, e.g., apple, pear, quince

positive-sense RNA (+RNA) RNA that can serve directly as messenger RNA (contrasts with negative-sense RNA)

post-transcriptional gene silencing (acronym PTGS) a mechanism for sequence-specific RNA degradation in plants, resulting in the turning off of a gene; used as a host plant defense against viruses by degrading viral RNAs created during replication

posterior toward the back or rear (contrasts with anterior)

powdery mildew a white, powdery, superficial ascomycetous fungus that is an obligate parasite and usually is found only on one or a few closely related species of plants

predispose (n. predisposition) to make prone to infection and disease

primary inoculum inoculum, usually from an overwintering source, that initiates disease in the field, as opposed to inoculum that spreads disease during the season (see also initial inoculum) (contrasts with secondary inoculum)

primary host sometimes used to refer to the telial host of a heteroecious rust fungus, and sometimes used to refer to the most economically important host

primary leaf first true leaf that emerges on a plant following the cotyledons

primary pollutant air pollutant that is released directly into the atmosphere and is harmful to plants, e.g., SO_2 or NO_x

primary root root that develops directly from the radicle of an embryo rather than from a crown or node

primer small fragment of nucleic acid with a free 3’-hydroxyl group necessary for initiation of DNA and, sometimes, RNA synthesis; often specific fragments chosen for use in polymerase chain reaction (PCR) for rapid identification of pathogens

proinhibitin a plant defense compound that is already present before invasion by a pathogen

prokaryote an organism without internal membrane-bound organelles, lacking a distinct nucleus, such as bacteria and mollicutes (contrasts with eukaryote)

promycelium (pl. promycelia) in rust and smut fungi, a germ tube issuing from the teliospore and bearing the basidiospores

propagative transmission pathogen transmission characterized by a long period of acquisition of the pathogen (usually a mollicute, e.g., phytoplasma or spiroplasma, and sometimes a virus) by a vector (typically an insect), a latent period before the vector is able to transmit the pathogen, and retention of the pathogen by the vector for a long period because the pathogen reproduces or replicates in the vector (see also circulative transmission, persistent transmission) (contrasts with nonpersistent transmission, stylet-borne transmission)

propagule any part of an organism capable of independent growth

protectant an agent, usually a chemical, applied to a plant surface in advance of a pathogen to prevent infection

protectant fungicide a fungicide that remains on the surface where it is applied; a fungicide with no after-infection activity (see also contact fungicide) (contrasts with systemic fungicide)

protection various methods of plant disease management, including cultural practices that create barriers or reduce the chance of infection, chemical protection, methods of biological control that protect plants, and genetic resistance

protein a nitrogen-containing organic compound composed of units called amino acids

protoplasm living contents of a cell

protoplast living cell exclusive of a wall

proximal nearest to the point of attachment (contrasts with distal)

pseudothecium (pl. pseudothecia) perithecium-like fruiting body containing asci and ascospores dispersed rather than in an organized hymenium; an ascostroma with a single locule or cavity and containing bitunicate asci

PTGS (acronym for p̲ost-t̲ranscriptional gene s̲ilencing) a mechanism for sequence-specific RNA degradation in plants, resulting in the turning off of a gene; used as a host plant defense against viruses by degrading viral RNAs created during replication

Puccinia pathway the region through which rust urediniospores move from southern areas through all grain-producing areas of the United States to Canada each season

pupa (pl. pupae; v. pupate) quiescent stage between the larva and the adult of certain insects

pustule small, blisterlike elevation of epidermis formed as spores emerge

pv. (abbrev. for pathovar) a subdivision of a plant-pathogenic bacterial species defined by host range; pathovar for bacteria is equivalent to *forma specialis* for fungi

pycnidiospore spore (conidium) produced in a pycnidium

pycnidium (pl. pycnidia) asexual, globose or flask-shaped fruiting body containing conidia

pycniospore haploid, sexually derived spore formed in a pycnium of a rust fungus (see also spermatium)

pycnium (pl. pycnia) globose or flask-shaped haploid fruiting body of a rust fungus bearing receptive hyphae and pycniospores (see also spermagonium)

pyramiding the addition, through plant breeding or genetic engineering, of several resistance genes into a single plant cultivar

qualitative resistance resistance reactions that can be placed in distinct categories, usually conferred by one or a few genes (contrasts with quantitative resistance)

quantitative resistance resistance reactions that have no distinct classes but vary continuously from resistant to susceptible, the result of few to many genes, the individual effects of which may be small and difficult to detect (contrasts with qualitative resistance)

quarantine legislative control of the transport of plants or plant parts to prevent the spread of pests or pathogens

quiescent dormant or inactive

quorum sensing the ability of bacteria to interact with each other through a variety of mechanisms; allows a population of bacteria to behave more like a multi-cellular organism

race subgroup or biotype within a species or variety, distinguished from other races by virulence, symptom expression, or host range, but not by morphology

rachis elongated main axis of an inflorescence

radicle part of the plant embryo that develops into the primary root

race-nonspecific resistance resistance that is effective against all biotypes of the pathogen (see also durable resistance, general resistance, horizontal resistance) (contrasts with specific resistance, vertical resistance)

ray tissue that extends radially in the secondary xylem and phloem of a woody plant

receptacle the structure of a flower that bears the reproductive organs

receptive hypha the part of a rust fungus pycnium (spermogonium) that receives the nucleus of a pycniospore (spermatium)

receptor a site that recognizes and binds an elicitor; any organ or molecular site that is sensitive to a distinct (specific) signal molecule

recessive describes a phenotypic trait that is expressed in diploid organisms only if both parents contribute the trait to the progeny (contrasts with dominant)

recombinant DNA DNA molecules in which sequences, not normally contiguous, have been placed next to each other by in vitro methods

reniform kidney-shaped

replicase an enzyme involved in the replication (copying) of nucleic acid

replication the process by which a DNA or RNA molecule makes an exact copy of itself; repetition of an experiment or procedure at the same time and place (one of several identical experiments, procedures, or samples); the process by which a virus particle induces the host cell to reproduce the virus

resin sticky to brittle plant product derived from essential oils; often a defense compound of conifers

resinosis a disease symptom in which there is a copious flow of resin from a wound or infection site of a conifer

resistant (n. resistance) possessing properties that prevent or impede disease development (contrasts with susceptible)

respiration series of chemical reactions that make energy available through oxidation of carbohydrates and fat

resting spore spore, often thick-walled, that can remain alive in a dormant state for some time, later germinating and capable of initiating infection

restriction endonuclease enzyme that cleaves DNA at a particular base sequence; sometimes informally referred to as a restriction enzyme

reticulate having netlike markings

reverse transcriptase enzyme used to make complementary DNA from a piece of RNA, such as a plant virus genome

Rhizobium genus of bacteria that live symbiotically with roots of leguminous plants; during the symbiosis, atmospheric nitrogen gas is converted into a form useable by the plant

Rhizobium nodules galls on roots caused by *Rhizobium* spp.

rhizome a mostly horizontal, jointed, fleshy, often elongated, usually underground stem

rhizomorph macroscopic ropelike strand of compacted tissue formed by certain fungi

rhizosphere microenvironment in the soil immediately around roots

rhizosphere-competent used to describe microorganisms adapted to living in the rhizosphere of a plant

ribonucleic acid (acronym RNA) several nucleic acids composed of repeating units of ribose (a sugar), a phosphate group, and a purine (adenine or guanine) or a pyrimidine (uracil or cytosine) base; transcribed from DNA and involved in translation to proteins

ribosomal RNA (abbrev. rRNA) RNA molecules forming part of the ribosomal structure

ribosome a subcellular protoplasmic particle, made up of one or more RNA molecules and several proteins, involved in protein synthesis

ringspot disease symptom characterized by yellowish or necrotic rings enclosing green tissue, as in some plant diseases caused by viruses

RNA (acronym for ribonucleic acid) several nucleic acids composed of repeating units of ribose (a sugar), a phosphate group, and a purine (adenine or guanine) or a pyrimidine (uracil or cytosine) base; transcribed from DNA and involved in translation to proteins

RNA interference (RNAi) a process within living cells in which a double-stranded complementary RNA targets a specific messenger RNA for destruction, blocking the function of (silencing) the gene from which the mRNA was transcribed

rogue to remove and destroy individual plants that are diseased, infested by insects, or otherwise undesirable

root cap a group of cells on a root that protects the growing tip

root exudate one of the various compounds that leak from growing and expanding sections of roots as well as from broken cells at exit points of lateral roots

root graft the fusion of roots of two adjacent plants so that their water- and food-conducting (vascular) systems become joined

root hair threadlike, single-celled outgrowth from a root epidermal cell

rootstock portion of the stem (trunk) and associated root system into which a bud or scion is inserted in grafting; fleshy overwintering part of a herbaceous perennial plant with buds and eyes (contrasts with scion)

rosette disease symptom characterized by short, bunchy growth habit resulting from shortened internodes and no comparable reduction in leaf size

rot softening, discoloration, and often disintegration of plant tissue as a result of fungal or bacterial infection

rotation growth of different kinds of crops in succession in the same field

roundworm nematode

rRNA(abbrev. for ribosomal RNA) RNA molecules forming part of the ribosomal structure

rugose wrinkled, roughened

runner a slender, horizontal stem that grows close to the soil surface (see also stolon)

runner plant a new plant produced asexually on a runner or stolon

russet brownish, roughened areas resulting from cork formation

rust a disease caused by a specialized group of basidiomycetes that often produces spores of a rusty color

sanitation destruction or removal of infected and infested plants or plant parts; decontamination of tools, equipment, containers, work space, hands, etc.

sap transmission transmission, usually of viruses, by rubbing sap from an infected plant onto a healthy plant to cause infection

saprobe (adj. saprobic) an organism that obtains nourishment from nonliving organic matter (see also saprophyte, saprotroph)

saprophyte (adj. saprophytic) an organism that obtains nourishment from nonliving organic matter (see also saprobe, saprotroph)

saprotroph (adj. saprotrophic) an organism that obtains nourishment from nonliving organic matter (see also saprobe, saprophyte)

sapwood physiologically active zone of wood contiguous to cambium (contrasts with heartwood)

SAR (acronym for systemic acquired resistance) a rapid and coordinated defense response against a variety of pathogens as a signal travels throughout the plant; a reduction in disease symptoms on a portion of a plant distant from the area where a hypersensitive response occurred or other stimulus was applied; (see induced systemic resistance)

scab roughened, crustlike diseased area on the surface of a plant organ

scald a necrotic condition in which tissue is usually bleached and has the appearance of having been exposed to high temperatures

scion portion of a shoot used for grafting onto the root-stock (contrasts with rootstock)

sclerenchyma (adj. sclerenchymatous) tissue made up of thick-walled plant cells

sclerotium (pl. sclerotia) a vegetative resting body of a fungus, composed of a compact mass of hyphae with or without host tissue, usually with a darkened rind

scorch any symptom that suggests the action of flame or fire on the affected part, often seen at the margins of leaves

secondary infection infection resulting from the spread of infectious material produced after a primary infection or from secondary infections without an intervening inactive period

secondary inoculum inoculum produced by infections that took place during the same growing season (contrasts with initial inoculum, primary inoculum)

secondary metabolite a compound produced in microbes (e.g., mycotoxins, syringomycins) or plants (e.g., caffeine, nicotine) that is not necessary for normal growth and development

secondary organism organism that multiplies in already diseased tissue but is not the primary pathogen

secondary pollutant air pollutant that must be chemically produced from other air pollutants, e.g., ozone (O_3), which is a product of a photochemical reaction of exhaust products from combustion engines in the atmosphere

secondary root branch from a primary root

sedentary remaining in a fixed location (contrasts with migratory)

seed ripened ovule consisting of an embryo and stored food enclosed by a seed coat

seed treatment application of a biological agent, chemical substance, or physical treatment to seed to protect the seed or plant from pathogens or to stimulate germination or plant growth

seedborne carried on or in a seed

selective medium a culture medium containing substances that specifically inhibit or prevent the growth of some species of microorganisms

self-fertilization a process in which sexual reproduction occurs as a result of the fusion of sex cells produced by the same individual (contrasts with cross-fertilization)

self-pollination transfer of pollen from the anthers to the stigma of the same flower (contrasts with cross-pollination)

senesce (adj. senescent, n. senescence) to decline, as with maturation, age, or disease stress

sepal one of the modified leaves comprising a calyx

septate with cross walls; having septa (contrasts with coenocytic, nonseptate)

septum (pl. septa; adj. septate) dividing wall; in fungi, cross wall

serology (adj. serologic) a method using specificity of the antigen-antibody reaction for the detection and identification of antigenic substances and the organisms that carry them

serrate edged with teeth, like a saw

sessile pertaining to a leaf, leaflet, flower, floret, fruit, ascocarp, basidiocarp, etc., without a stalk, petiole, pedicel, stipe, or stem; (of nematodes) permanently attached; not capable of moving about

seta (pl. setae) bristle or hairlike structure, usually deep yellow or brown and thick-walled

severity values a means to quantify accumulating opportunities for pathogen infection (e.g., infection periods), with reference to a predetermined threshold that, when met, triggers a disease-management activity (e.g., a fungicide application); used in disease prediction or forecasting

sexual reproduction reproduction involving fusion of two haploid nuclei (karyogamy) to form a diploid nucleus, followed by meiosis (reduction division) back to haploid nuclei at some point in the life cycle, resulting in genetic recombination (contrasts with asexual reproduction)

sexual spore spore produced during the sexual cycle

sexually compatible able to be cross-mated or cross-fertile

shepherd's crook the curved, apical portion of a blighted stem (see also crozier)

shot-hole symptom in which small lesions fall out of leaves, giving the leaf the appearance of being hit by buckshot

sieve element (syn. sieve tube element) a tube-shaped living cell in the phloem, functioning in the transport of dissolved organic substances in the plant

sign indication of disease from direct observation of a pathogen or its parts (contrasts with symptom)

single gene resistance resistance conferred by a single gene (see also monogenic resistance) (contrasts with multigenic resistance, oligogenic resistance, polygenic resistance)

slime molds a group of nonpathogenic fungus-like organisms that form a vegetative amoeboid plasmodium and spores (see also myxomycetes)

smut a disease caused by a smut fungus (Ustilaginales) in the Basidiomycota or the fungus itself, characterized by masses of dark brown or black, dusty to greasy teliospores that generally accumulate in black, powdery sori

soft rot softening, discoloration, and often disintegration of plant tissue as a result of fungal or bacterial infection

soil drench application of a solution or suspension of a chemical to the soil, especially application of a pesticide to manage soilborne pathogens

soilborne carried on or beneath the soil surface

soil inhabitant an organism that maintains its population in soil over an extended period of time

soil invader an organism whose population in soil diminishes in several months to years

soil pasteurization process used to free soil of selected harmful microorganisms using heat

soil sterilization process used to free soil of all microorganisms

solarization a disease management practice in which soil is covered with polyethylene sheeting and exposed to sunlight, thereby heating the soil and reducing populations of soilborne plant pathogens

sooty mold black, nonparasitic, superficial fungal growth on honeydew produced by aphids and other phloem-feeding insects

sorus (pl. sori) compact fruiting structure, especially the erumpent spore mass in the rust fungi (Uredinales) and smut fungi (Ustilaginales); occasionally a group of fruiting bodies as in Synchytriaceae; a cluster of sporangia on a fern sporophyte

sp. (pl. spp.) abbreviation for species; a genus name followed by sp. means that the particular species is undetermined; spp. after a genus name means that several species are being referred to

species any one kind of life subordinate to a genus but above a race; a group of closely related individuals of the same ancestry, resembling one another in certain inherited characteristics of structure and behavior and relative stability in nature; the individuals of a species ordinarily interbreed freely and maintain themselves and their characteristics in nature

specific epithet the second word in a Latin binomial

specific resistance resistance that is effective against some biotypes or races of the pathogen, but not others, usually inherited monogenically and expressed qualitatively (see also vertical resistance) (contrasts with durable resistance, general resistance, horizontal resistance, race-specific resistance)

spermagonium (pl. spermagonia) structure in which male reproductive cells are produced; in rust fungi, globose or flask-shaped haploid fruiting body composed of receptive hyphae and spermatia (pycniospores) (see also pycnium for rust fungi)

spermatium (pl. spermatia) a male sex cell; a nonmotile male gamete; a haploid male gamete (see also pycniospore for rust fungi)

spicule copulatory organ of a male nematode

spike botanical term for an unbranched inflorescence in which flowers (or spikelets of grasses) are attached directly, without petioles, to a central stem, e.g., wheat, gladiolus

spikelet spikelike appendage composed of one or more reduced flowers and associated bracts; unit of inflorescence in grasses; a small spike

spiroplasma spiral-shaped, plant-pathogenic mollicute (prokaryote without cell wall)

spontaneous generation, theory of the theory, now known to be invalid, that plants, animals, and microorganisms arise from nonliving materials under certain environmental conditions

sporangiophore sporangium-bearing body of a fungus

sporangiospore nonmotile, asexual spore that is borne in a sporangium

sporangium (pl. sporangia) saclike fungal structure in which the entire contents are converted into an indefinite number of asexual spores

spore reproductive structure of fungi and some other organisms, containing one or more cells; a bacterial cell modified to survive an adverse environment

sporidium (pl. sporidia) basidiospore of rust fungi, smut fungi, and other Basidiomycota

sporocarp spore-bearing fruiting body

sporodochium (pl. sporodochia) superficial, cushion-shaped asexual fruiting body consisting of a cluster of conidiophores

sporophore a spore-producing or spore-bearing structure such as a conidiophore, conidioma, ascoma/ascocarp, or basidioma/basidiocarp

sporophyte the diploid stage of a plant (see gametophyte)

sporulate to produce spores

spot a symptom of disease characterized by a limited necrotic area, as on leaves, flowers, and stems

springwood part of the yearly xylem growth ring in woody plants formed early in the growing season, consisting typically of cells that are larger than those formed later in the season (contrasts with summerwood)

stabilizing selection the theorized competitive disadvantage of unnecessary virulence genes; races with excess genes would have decreased fitness relative to races with fewer virulence genes, so a "super-race" would be less likely to appear in multiline crops

staghead defoliated, dead, or dying major branches in the crown of a tree, usually resulting from inadequate water uptake or translocation

stamen (adj. staminal) male structure of a flower, composed of a pollen-bearing anther and a filament, or stalk

stele central cylinder of vascular tissue (especially in roots)

stem pitting a disease symptom characterized by depressions on the stem

sterigma (pl. sterigmata) small, usually pointed projection that supports a spore

sterile unable to reproduce sexually; free of living microorganisms

sterile fungus a fungus that is not known to produce any kind of spores

sterilization (adj. sterilized) the total destruction of living organisms by various means, including heat, chemicals, or irradiation

stigma portion of a flower that receives pollen and on which the pollen germinates

stipe stalk

stippling series of small dots or speckles in which chlorophyll is absent

stipule small, leaflike appendage at the base of a leaf petiole, usually occurring in pairs

stolon a slender, horizontal stem that grows close to the soil surface; in fungi, a hypha that grows horizontally along the surface (see also runner)

stoma (also stomate; pl. stomata; adj. stomatal) structure composed of two guard cells and the opening between them in the epidermis of a leaf or stem, functioning in gas exchange

stone fruit fruit with a stony endocarp, e.g., cherry, peach, plum

strain a distinct form of an organism or virus within a species, differing from other forms of the species biologically, physically, or chemically

Straminipila (Stramenopila) a kingdom including the Oomycota or oomycetes, along with brown and golden algae and diatoms. “Stramenopila” is the originally proposed spelling, whereas “Straminipila” has been proposed as more correct in form on the basis of the Latin root of the term

striate (n. striations) marked with delicate lines, grooves, or ridges

stroma (pl. stromata) compact mass of mycelium (with or without host tissue) that supports fruiting bodies or in which fruiting bodies are embedded

stunting reduction in height of a vertical axis resulting from a progressive reduction in the length of successive internodes or a decrease in their number

style slender part of many pistils located between the stigma and the ovary and through which the pollen tube grows

stylet stiff, slender, hollow feeding organ of plant-parasitic nematodes or sap-sucking insects, such as aphids or leafhoppers

stylet knob structure at the base of a nematode stylet (see also basal knob)

stylet-borne transmission a type of virus transmission in which the virus is acquired and transmitted by the vector after short feeding times and is retained by the vector for only a short period of time (see also nonpersistent transmission) (contrasts with circulative transmission, persistent transmission, propagative transmission)

suberize to convert into cork tissue

subgenomic RNA a piece of viral RNA, shorter than the entire genome of the virus, found in cells infected by the virus and sometimes encapsidated

subspecies a subpopulation of a species, defined on the basis of more than one character (morphologic for many organisms) that distinguishes the members of the subpopulation from other members of that species

substrate the substance on which an organism lives or from which it obtains nutrients; chemical substance acted upon, often by an enzyme

sulfur dioxide (SO_2) a primary air pollutant produced in industrial processes and coal burning that causes interveinal necrosis on broadleaved plants and tip necrosis on conifers

summerwood part of the yearly xylem growth ring in woody plants formed late in the growing season and consisting of cells smaller than those of springwood (contrasts with springwood)

sunscald or **sunburn** injury of plant tissues burned or scorched by direct sun

suppressive soil soil in which various diseases are naturally at lower levels than expected because of biological factors in the soil; an example of natural biological control

suscept an abbreviated term for a susceptible plant

susceptible (n. susceptibility) prone to develop disease when infected by a particular pathogen (contrasts with resistant)

symbiosis (adj. symbiotic; n. symbiont) mutually beneficial association of two different kinds of organisms

sympodial pertaining to proliferation of axes, in which each successive spore or branch develops behind and to one side of the previous apex, where growth has ceased

symptom indication of disease by reaction of the host, e.g., canker, leaf spot, wilt (contrasts with sign)

symptomless carrier a plant that, although infected with a pathogen (usually a virus), produces no obvious symptoms

syncytium (pl. syncytia) a multinucleate structure in root tissue formed by dissolution of common cell walls induced by secretions of certain sedentary plant-parasitic nematodes, e.g., cyst nematodes

synergism (adj. synergistic) greater than additive effect of interacting factors (contrasts with antagonism)

synnema (pl. synnemata) a headlike cluster formed by compact or fused, generally upright conidiophores, with branches and spores (see also coremium)

systematics the study of biological diversity and the evolutionary relationships between organisms

systemic pertaining to a disease in which the pathogen (or a single infection) spreads generally throughout the plant; pertaining to chemicals that spread internally through the plant

systemic acquired resistance (acronym SAR) a rapid and coordinated defense response against a variety of pathogens as a signal travels throughout the plant; a reduction in disease symptoms on a portion of a plant distant from the area where a hypersensitive response occurred or other stimulus was applied; (see induced systemic resistance)

systemic fungicide a fungicide that is absorbed into plant tissue and may offer some curative or after-infection activity; includes fungicides that are locally systemic, xylem-mobile (upward moving), and amphimobile (move in phloem upward as well as downward in the plant) (contrasts with contact fungicide, protectant fungicide)

taproot primary root that grows vertically downward and from which smaller lateral roots branch

taxonomy (adj. taxonomic) the science dealing with naming and classifying organisms

teleomorph the sexual form in the life cycle of a fungus (see also perfect state) (contrasts with anamorph, holomorph, imperfect state)

teliospore (sometimes called teleutospore, teleutosporodesm) thick-walled resting or overwintering spore produced by the rust fungi (Uredinales) and smut fungi (Ustilaginales) in which karyogamy occurs; it germinates to form a promycelium (basidium) in which meiosis occurs

telial host the host plant on which a heteroecious rust fungus produces telia and, sometimes, uredinia (contrasts with aecial host)

telium (pl. telia) fruiting body (sorus) of a rust fungus that produces teliospores

temporary wilt wilt caused by insufficient soil water from which a plant can recover when water is supplied

teratogen a chemical that causes malformations in the fetus

testa (pl. testae) seed coat

thallus vegetative body of a fungus

thatch tightly intertwined layer of plant litter from accumulations of undecomposed or partially decomposed plant residues

thermotherapy use of heat to reduce or eliminate pathogens in plant tissue; often used on plants prior to meristem culture to produce pathogen-free plants

thorax insect body part between the head and abdomen

tillage the process of turning or stirring the soil

tiller a lateral shoot, culm, or stalk arising from a crown bud; common in grasses

tissue group of cells, usually of similar structure, that perform the same or related functions

tissue culture in vitro method of propagating healthy cells from plant tissues

titer concentration of a virus

tolerance (adj. tolerant) ability of a plant to endure an infectious or noninfectious disease, adverse conditions, or chemical injury without serious damage or yield loss; (of pesticides) the amount of chemical reside legally permitted on an agricultural product entering commercial channels, usually measured in parts per million (ppm)

totipotency the ability of a cell to regenerate into any tissue or into an entire plant, because even a specialized cell contains all of the genetic information for necessary for an organism's development

toxicity capacity of a substance to interfere with the vital processes of an organism

toxin poisonous substance of biological origin

tracheid elongated conducting cell of the xylem, with tapering or oblique end walls and pitted walls

transcription the production of a complementary strand of RNA from a segment of DNA

transduction the transfer of genes from one organism to another by viruses, especially in bacteria

transfer RNA (abbrev. tRNA) the RNA that moves amino acids to the ribosome to be placed in the order prescribed by the messenger RNA

transformation the transfer of genetic materials from one organism to another by humans (genetic engineering); a means of genetic variation in bacteria by absorption and incorporation of DNA from another bacterial cell

transgenic possessing a gene from another species; used to describe the organisms that have been the subject of genetic engineering (see also genetically modified organism, GMO)

translation the assembling of amino acids into a protein using messenger RNA, ribosomes, and transfer RNA

translocation movement of water, nutrients, chemicals, or food materials within a plant

translucent so clear that light may pass through

transmit (n. transmission) to spread or transfer, as in spreading an infectious pathogen from plant to plant or from one plant generation to another

transpiration water loss by evaporation from leaf surfaces and through stomata

trap crop crop planted around a field to protect the inner crop from diseases transmitted by aerial vectors; host crop of a parasitic plant, such as witchweed (*Striga* spp.), that is planted to stimulate seed germination and later sacrificed by plowing under before the parasitic plant produces new seeds

trenching physical separation of soil in a vertical plane to sever grafted roots between trees

trichogyne a female receptive hypha

trichome a plant epidermal hair, of which several types exist

triplet codon a set of three nucleotide bases in DNA or RNA that code for an amino acid

tRNA (abbrev. for transfer RNA) the RNA that moves amino acids to the ribosome to be placed in the order prescribed by the messenger RNA

tuber an underground stem adapted for storage, typically produced at the end of a stolon

turgidity state of being rigid or swollen as a result of internal water pressure

tumor abnormal swelling or localized outgrowth, often roughly spherical, produced by a plant as a result of attack by a fungus, bacterium, nematode, insect, or other organism (see also gall)

tylosis (pl. tyloses) balloonlike extrusions of parenchyma cells into lumina of contiguous vessels that partially or completely block them

type the example on which the description of a scientific name is based and which fixes the application of the name (the type genus of a family, or the type species of a genus)

ultrastructure submicroscopic structure of a macromolecule, cell, or tissue

unicellular one-celled (contrasts with multicellular)

uniflagellate having one flagellum

unitunicate having a single ascus wall (contrasts with bitunicate)

uninucleate having one nucleus (contrasts with multi-nucleate)

urediniospore (also urediospore, uredospore) the asexual, dikaryotic, often rust-colored spore of a rust fungus, produced in a structure called a uredinium; the "repeating stage" of a heteroecious rust fungus, i.e., capable of infecting the host species on which it is produced

uredinium (also uredium; pl. uredinia) the fruiting body (sorus) of a rust fungus that produces urediniospores

vacuole generally spherical organelle within a plant cell, bound by a membrane and containing dissolved materials such as metabolic precursors, storage materials, or waste products

variegation pattern of two or more colors in a plant part, as in a green and white leaf

variety (adj. varietal) a plant type within a species, resulting from deliberate manipulation, that has recognizable characteristics (color, shape of flowers, fruits, seeds, height, and form) (see also cultivar)

vascular pertaining to fluid-conducting (xylem and phloem) tissues in plants

vascular bundle strand of conductive tissue, usually composed of xylem and phloem (in leaves, small bundles are called veins)

vascular cylinder the cylinder of vascular tissue in stems or roots (see also stele)

vascular wilt disease a xylem disease that disrupts normal uptake of water and minerals, resulting in wilting and yellowing of foliage

vector a living organism (e.g., insect, mite, bird, higher animal, nematode, parasitic plant, human) able to carry and transmit a pathogen and disseminate disease; in genetic engineering, a vector or cloning vehicle is a self-replicating DNA molecule, such as a plasmid or virus, used to introduce a fragment of foreign DNA into a host cell

vegetative referring to somatic or asexual parts of a plant that are not involved in sexual reproduction

vegetative propagation asexual reproduction; in plants, the use of cuttings, bulbs, tubers, and other vegetative plant parts to grow new plants

vein small vascular bundle in a leaf

vein banding symptom of virus disease in which regions along veins are either darker green or distinctly more yellow than tissue between veins

vein clearing disappearance of green color in or around leaf veins

vermiform worm-shaped

vertical resistance resistance that is effective against some biotypes or races of the pathogen, but not others, usually inherited monogenically and expressed qualitatively (see also specific resistance) (contrasts with general resistance, horizontal resistance, race-nonspecific resistance)

Vertifolia effect the loss of general (durable, horizontal, host-nonspecific) resistance in a cultivar after several generations of selection during which a major gene confers resistance to the dominant race or biotype of the pathogen; first observed in the potato cultivar Vertifolia with late blight resistance

vesicle thin sac in which zoospores are differentiated and released; the bulbous head terminating the conidiophores of *Aspergillus*; structure formed by endomycorrhizal fungi within living cells of the root

vessel water-conducting structure of xylem tissue with pit openings in end walls

viable (n. viability) the state of being alive; able to germinate, as seeds, fungal spores, sclerotia, etc.; capable of growth

virescence (adj. virescent) state or condition in which normally white or colored tissues (e.g., flower petals) become green

virion complete virus particle

viroid an infectious, nonencapsidated (naked) circular, single-stranded RNA

viroplasm cellular inclusions that are sites of synthesis of viral components and the assembly of virus particles

virulence degree or measure of pathogenicity; relative capacity to cause disease

virulent highly pathogenic; having the capacity to cause severe disease (contrasts with avirulent)

viruliferous virus-laden, usually applied to insects or nematodes as vectors

virus a submicroscopic, intracellular, obligate parasite consisting of a core of infectious nucleic acid (either RNA or DNA) usually surrounded by a protein coat

viscin a sticky tissue produced on the seed coat of parasitic flowering plants in the Viscaceae (mistletoes) that helps attach the seed to the host plant branch

volunteer self-set plant; plant seeded by chance

vulva exterior opening of a mature female nematode's reproductive system

walling-off separation of diseased from healthy tissues by barrier tissues produced by a diseased plant

water-soaked a disease symptom in which plant tissues or lesions appear wet, dark, and usually sunken and translucent

water sprout small, rapidly growing shoot or branch on a large stem, developed from adventitious tissues

white rot (of wood) type of wood decay resulting from enzymatic action of fungi; it degrades all components of wood, including lignin, leaving the wood light-colored and spongy

white rust common name usually used for diseases caused by oomycetes in the genus *Albugo*

whorl circular arrangement of like parts

wild type the phenotype characteristic of the majority of individuals of a species under natural conditions

wilt drooping of leaves and stems from lack of water (inadequate water supply or excessive transpiration); vascular disease that interrupts normal water uptake

wind break a row or other grouping of trees or shrubs used to provide protection against the effects of high-velocity winds

winterburn foliar necrosis, often marginal, of plants that retain their leaves in winter, resulting from water deficiency because they cannot take up water from frozen soils

witches' broom disease symptom characterized by an abnormal, massed, brushlike development of many weak shoots arising at or close to the same point

wood secondary xylem

wound injuries to plant tissues that often breach barriers (cuticle, bark, cell walls) that might otherwise exclude pathogens; some pathogens (e.g., viruses) can enter plants only through a wound; wounds may occur from natural growth processes, physical and chemical agents, animals (especially insects), and many human agricultural activities, such as pruning

xylem water- and mineral-conducting, food-storing, supporting tissue of a plant

XLB (acronym for xylem-limited fastidious bacteria) bacterial pathogens of plants found only in the xylem, causing wilt, scorch, and stunting symptoms; vectored by insects that feed on xylem fluid; not sap transmissible; require complex culture media

xylem-limited fastidious bacteria (acronym XLB) bacterial pathogens of plants found only in the xylem, causing wilt, scorch, and stunting symptoms; vectored by insects that feed on xylem fluid; not sap transmissible; require complex culture media

yeast unicellular ascomycetous fungus that reproduces asexually by budding

yellows disease characterized by chlorosis and stunting of the host plant

yield an aggregate of the products resulting from growth or cultivation

zonate pertaining to a targetlike development of a tree canker, characterized by successive, perennial rings of callus; referring to any symptom appearing in concentric rings

zoosporangium (pl. zoosporangia) sporangium, or spore case, that bears zoospores

zoospore spore with flagella, capable of locomotion in water

zygomycetes a fungus group, also called the Zygomycota, characterized by nonseptate hyphae, sexual zygospores, and sporangiospores produced in a sporangium; common postharvest pathogens

zygospore sexual resting spore formed from the union of gametangia in the zygomycetes

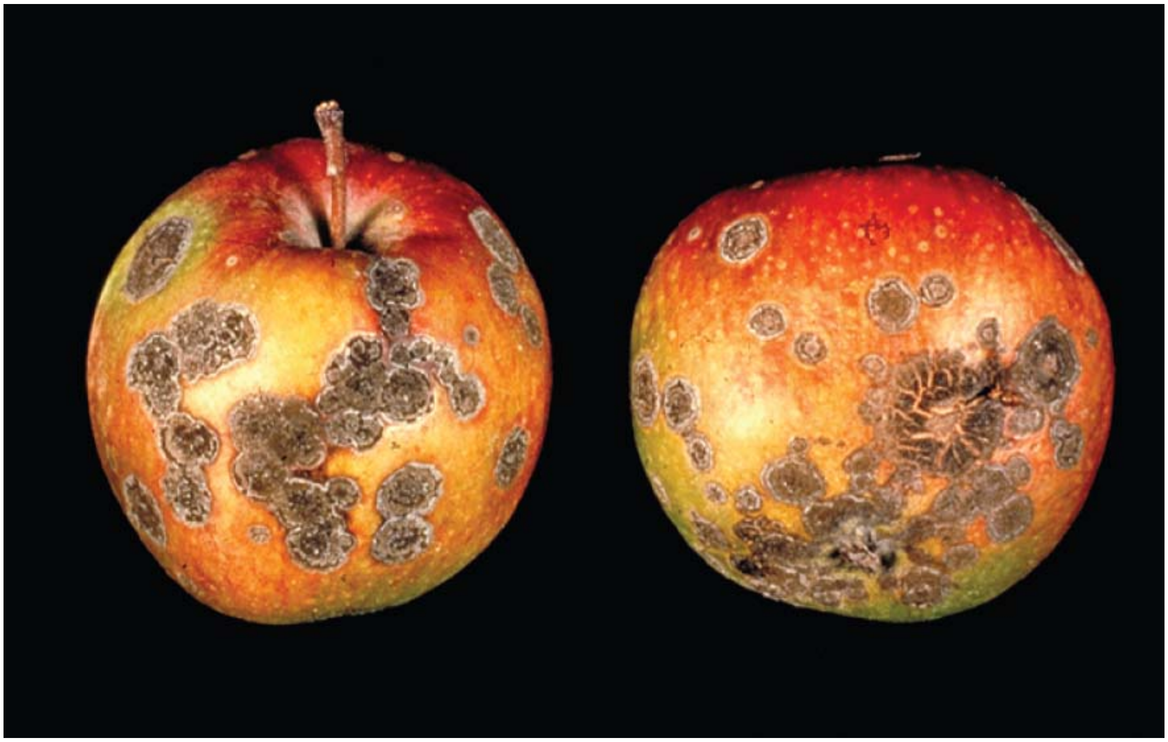


Figure 1.1.
Apple scab symptoms on fruit.

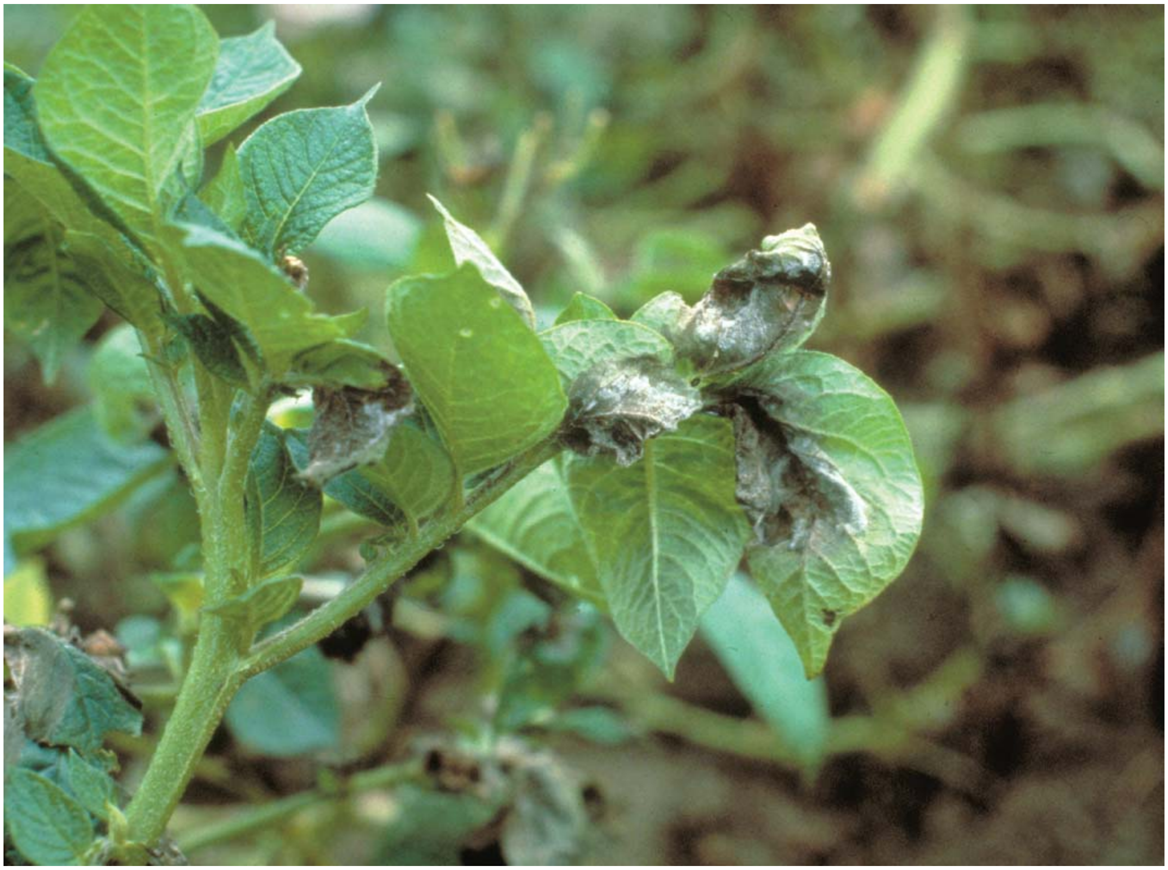


Figure 1.2.
Potato plant with late blight.

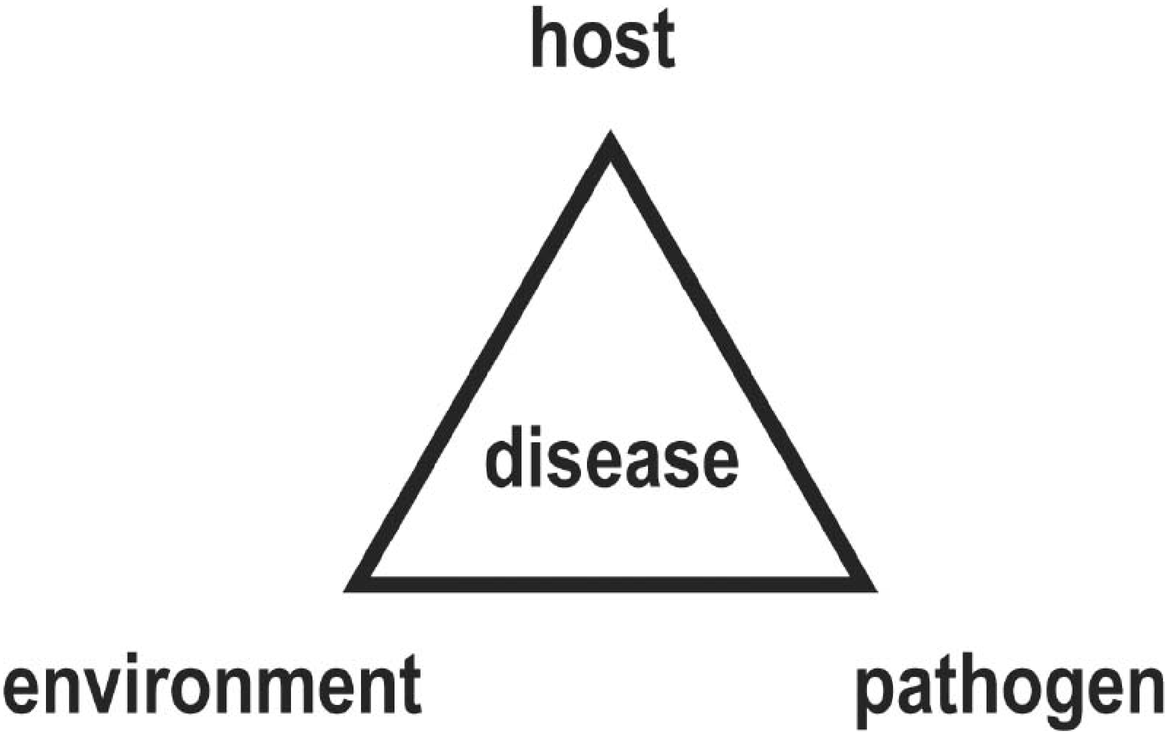
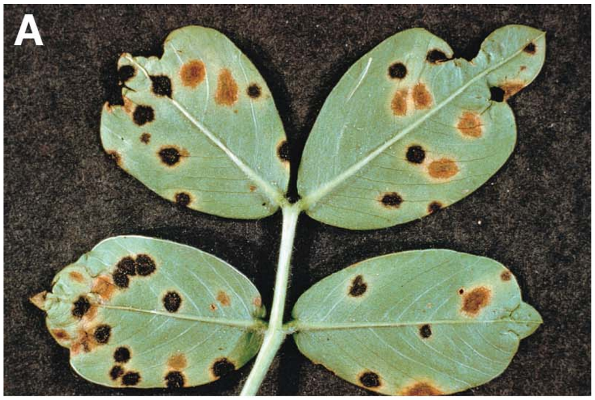
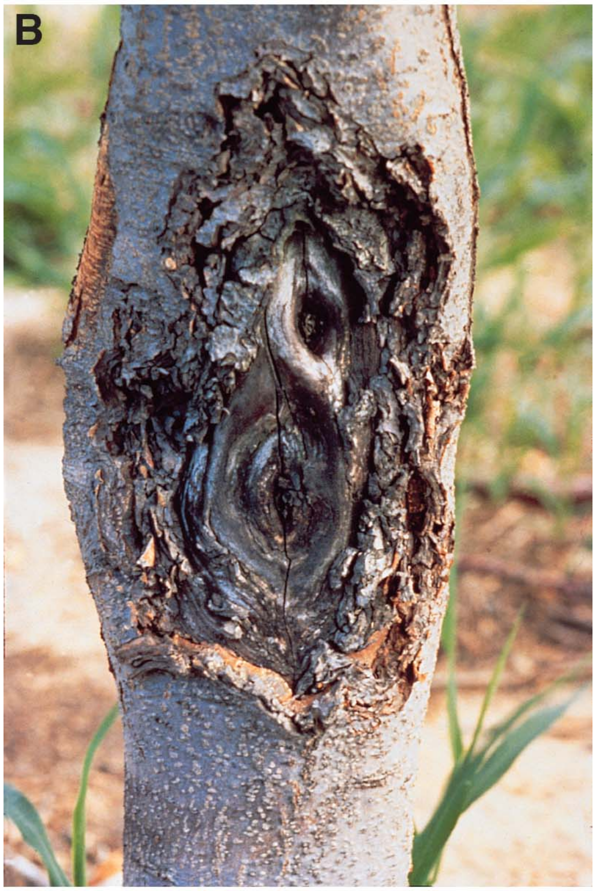

host
disease
environment
pathogen

Figure 1.3.
The disease triangle.


A


B

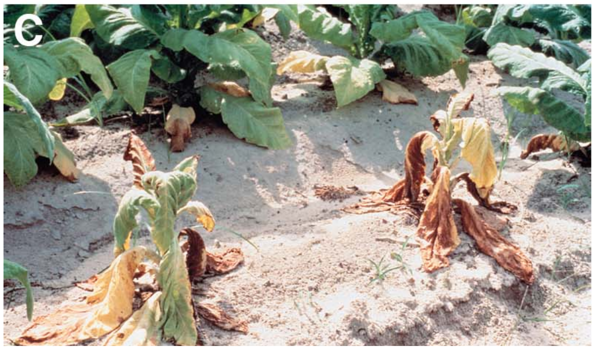

C

Figure 1.4.
Common symptoms of plant diseases: **A**, leaf spot; **B**, canker; **C**, wilt.

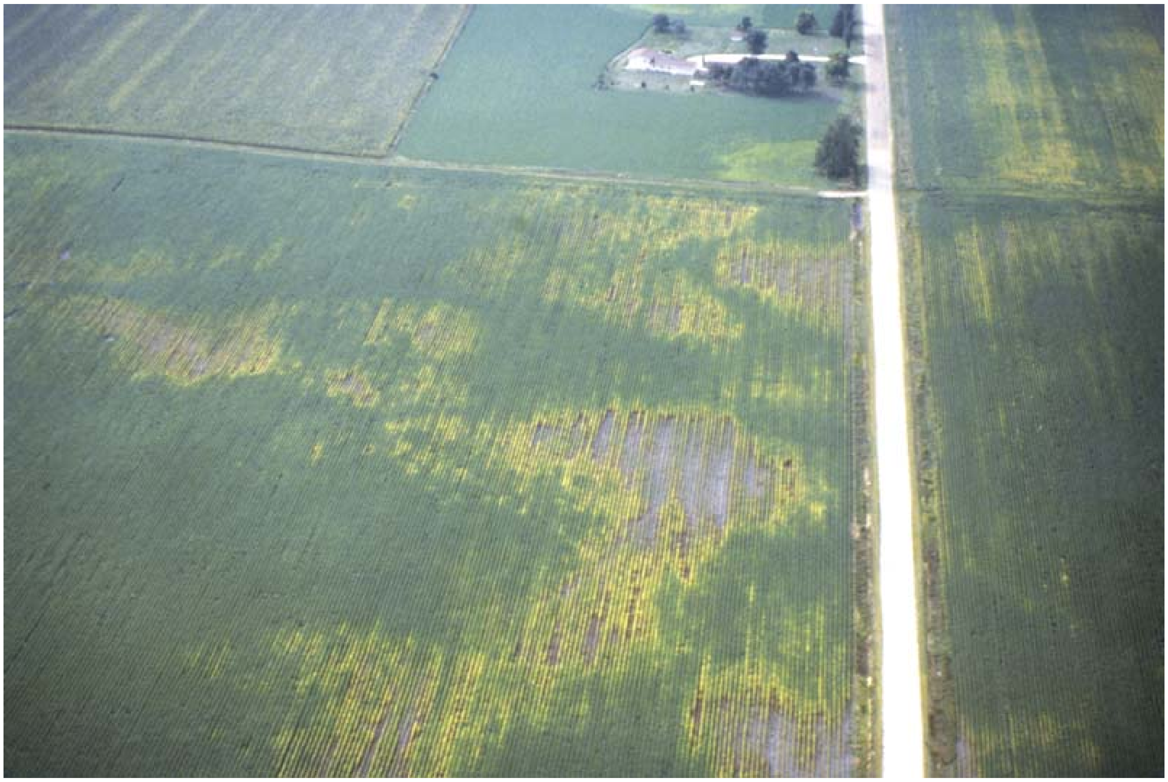


Figure 1.5.
Circular pattern of damage caused by the soybean cyst nematode. Such patterns are typical of diseases caused by soilborne pathogens.

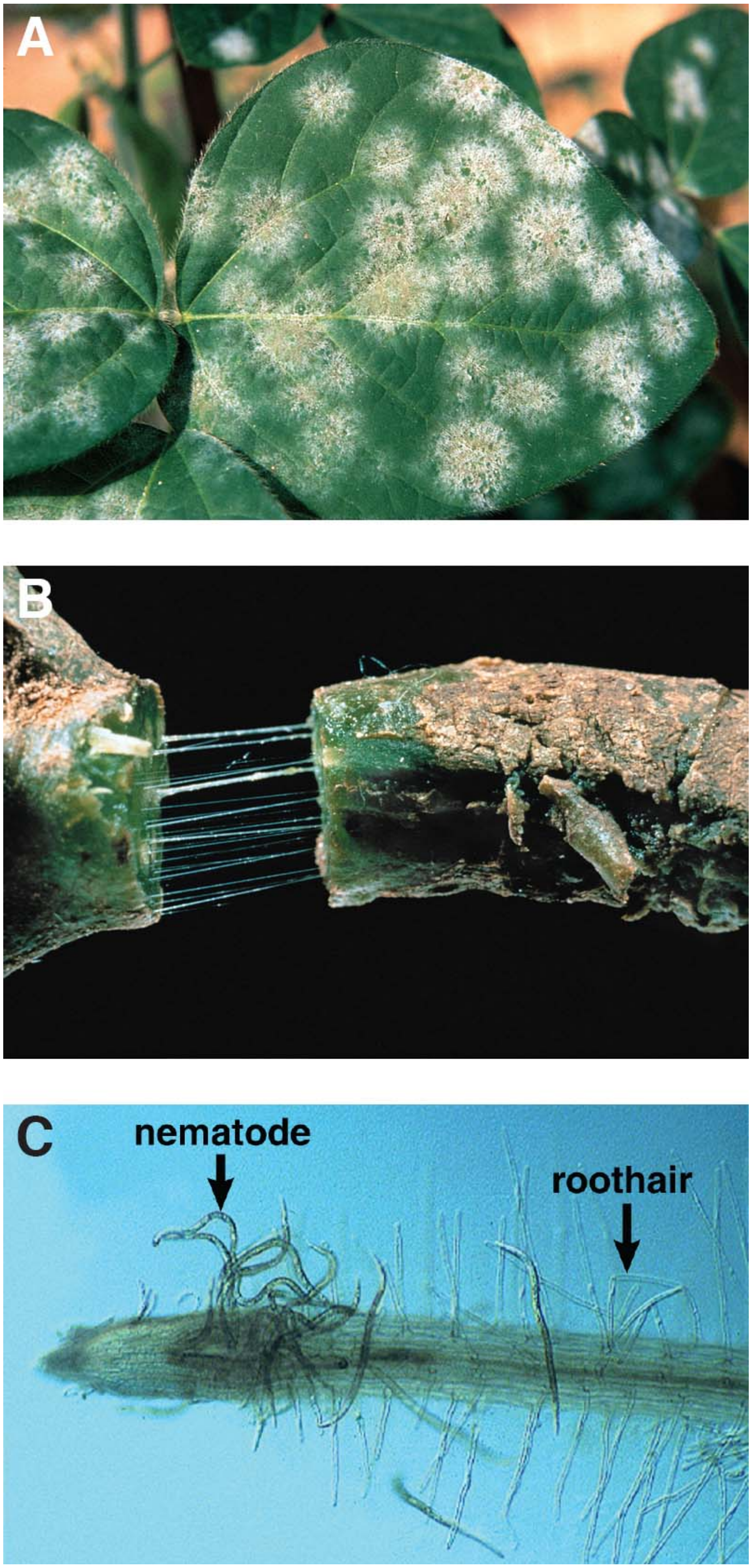
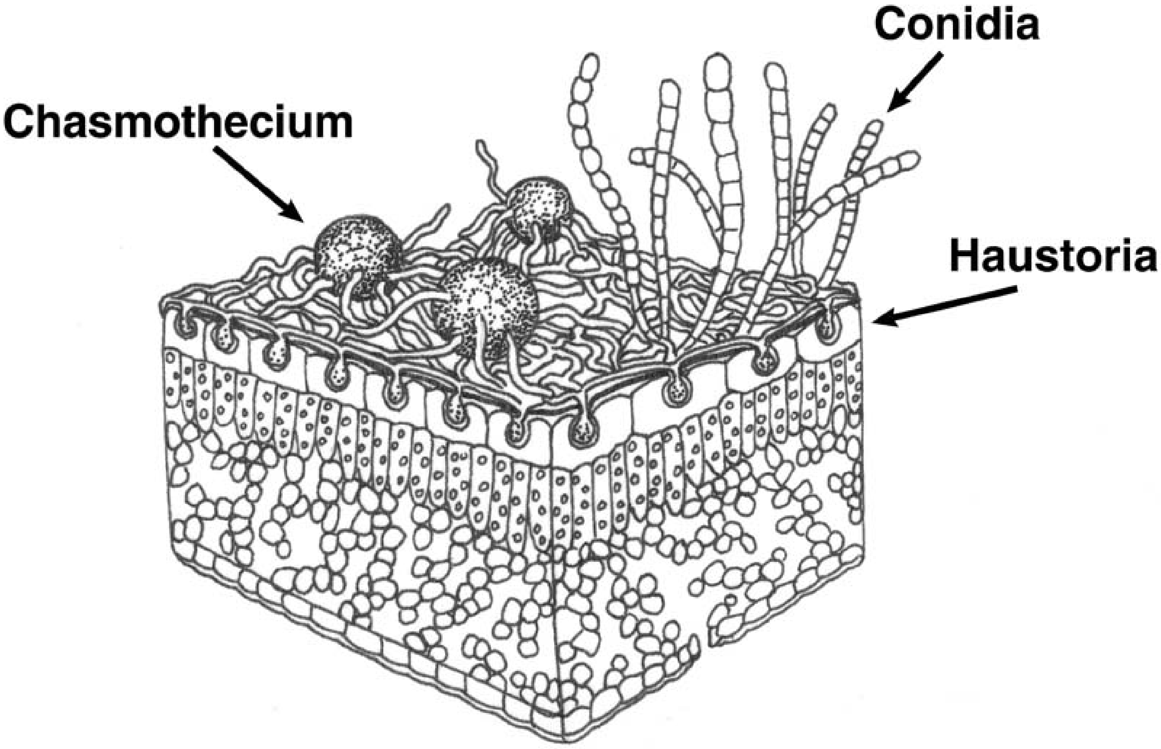

A
B
C
nematode
roothair

Figure 1.6.
Common signs of pathogens: **A**, mycelium and spores of a powdery mildew fungus; **B**, bacterial ooze; **C**, nematodes on a root.



Conidia
Chasmothecium
Haustoria

Figure 1.7.
A powdery mildew fungus, a biotroph, on a leaf surface.

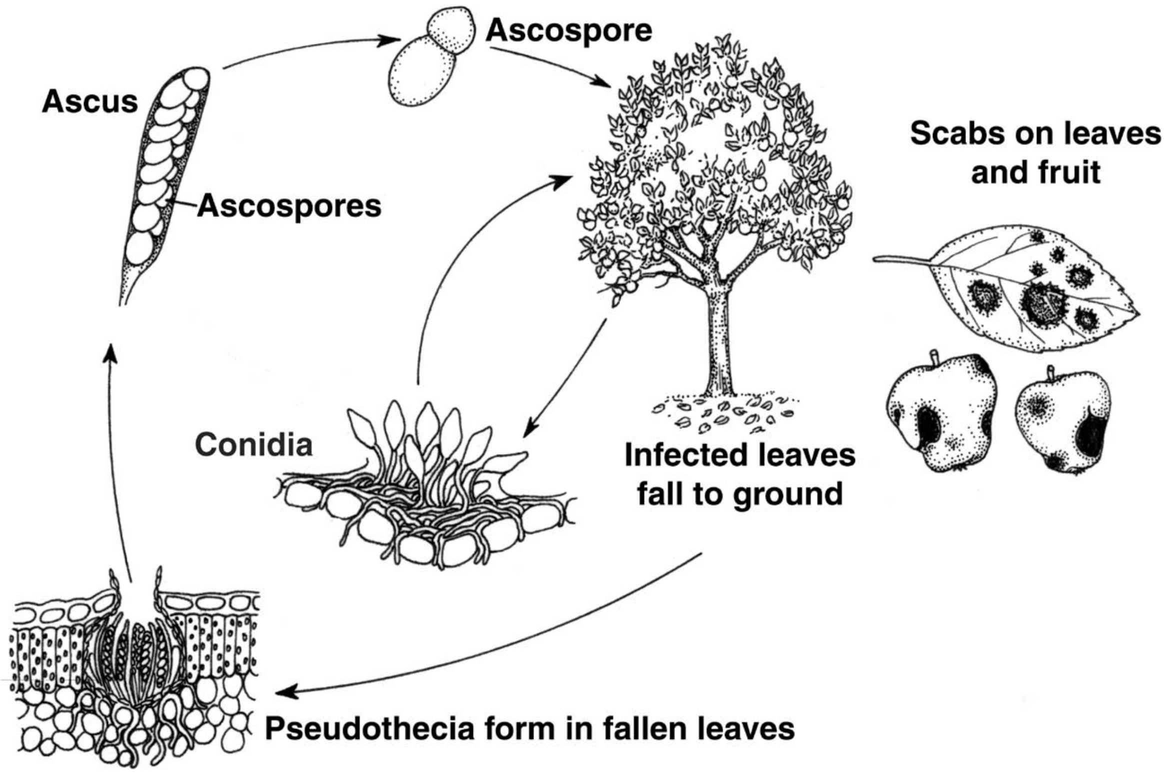

Ascospore
Ascus
Ascospores
Scabs on leaves and fruit
Conidia
Infected leaves fall to ground
Pseudothecia form in fallen leaves

Figure 1.8.
Disease cycle of apple scab.

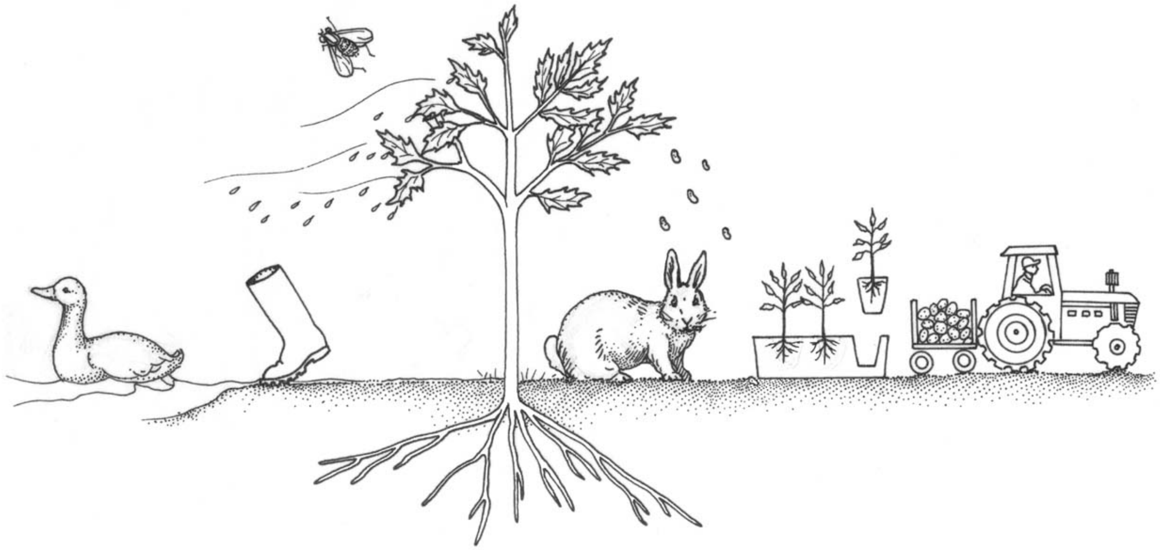


Figure 1.9.
Some common means of pathogen dispersal: insect vectors, tools and equipment, vegetative propagation, wind, and water.

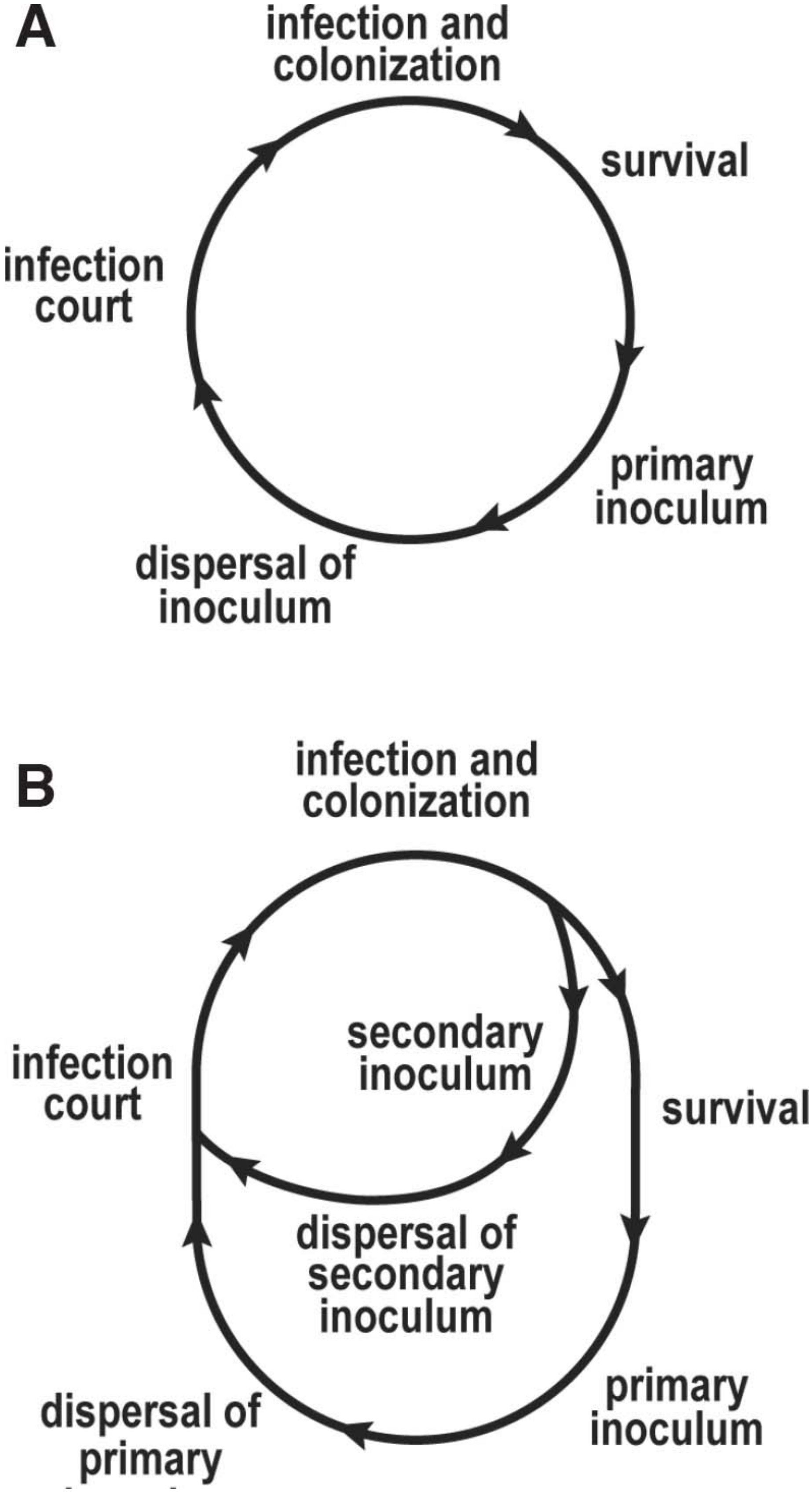

A
infection and colonization
survival
primary inoculum
dispersal of inoculum
infection court
B
infection and colonization
secondary inoculum
survival
infection court
dispersal of secondary inoculum
primary inoculum
dispersal of primary

Figure 1.10.

A, Monocyclic disease. **B**, Polycyclic disease.

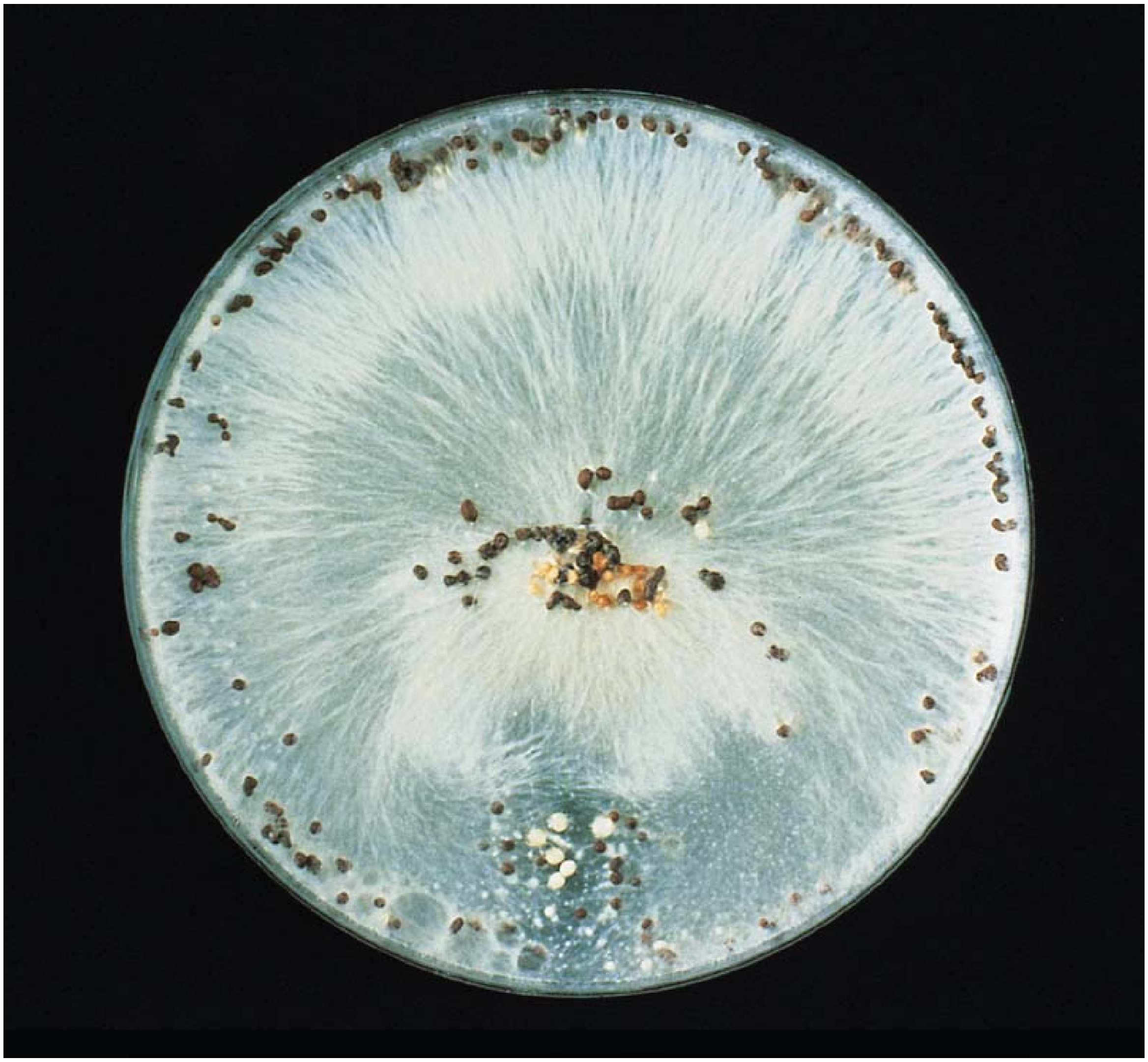


Figure 2.1.
Mycelium and sclerotia (survival structures) of the fungus *Sclerotium rolfsii* on potato-dextrose agar.

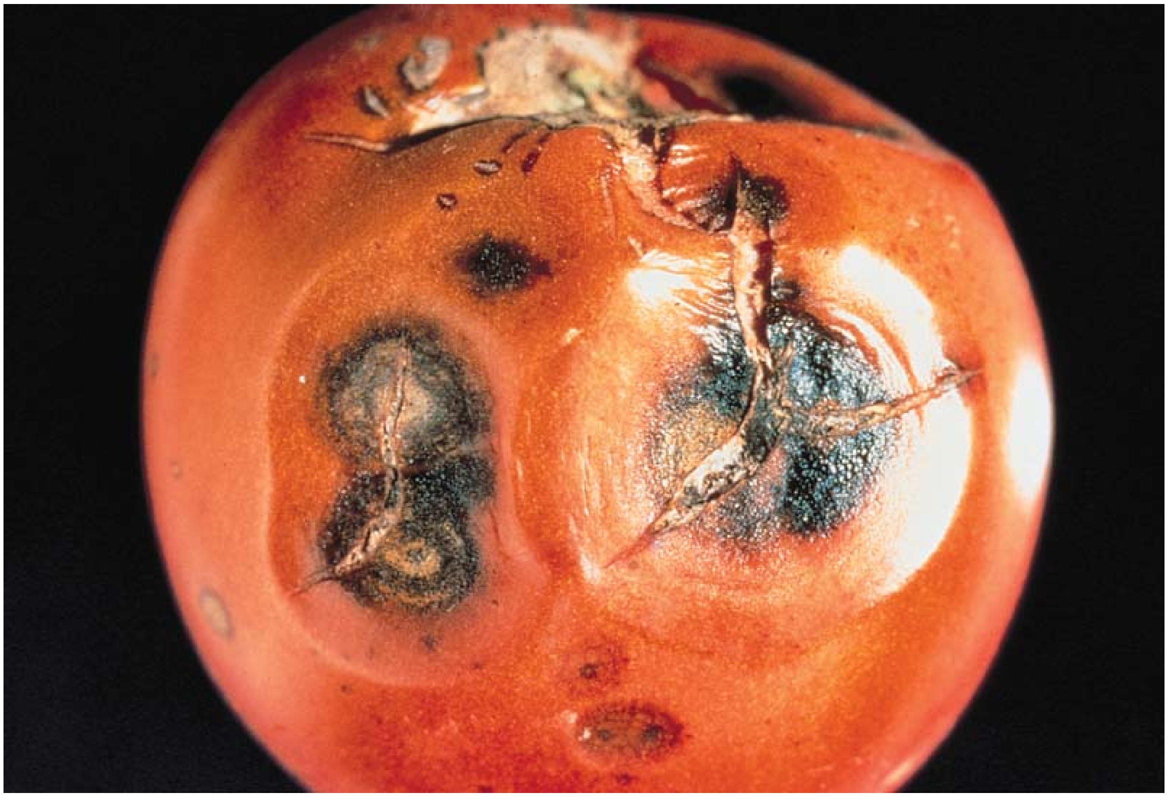


Figure 2.2.
Tomato with anthracnose. The round spots of infection are typical of a fungal disease.

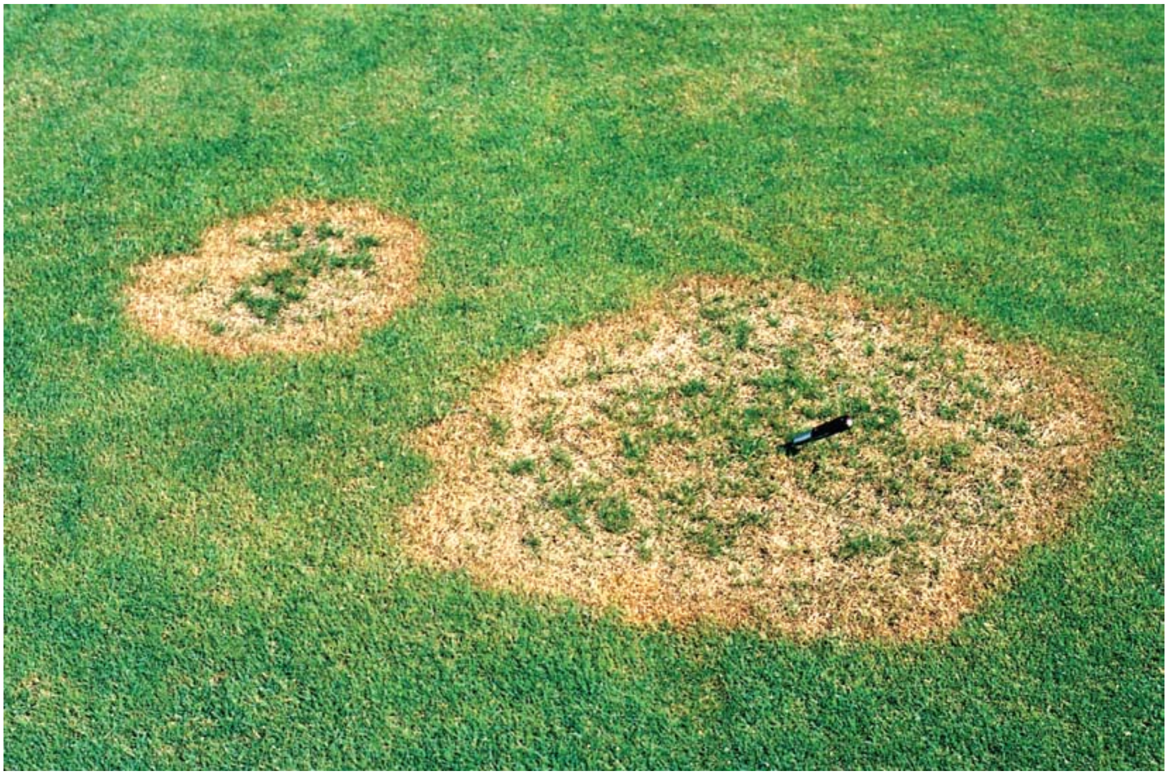


Figure 2.3.
Take-all patch of bentgrass. The affected areas are circular, typical of fungal diseases.

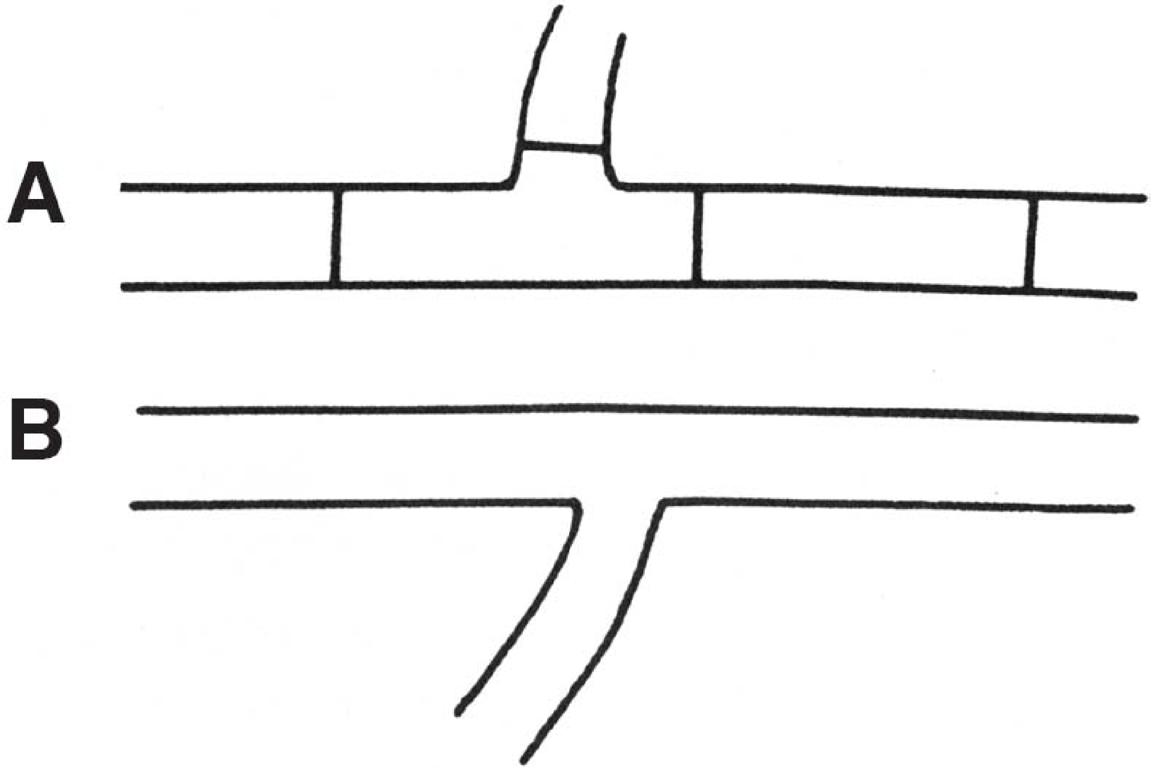


Figure 2.4.
Septate (**A**) and nonseptate hyphae (**B**). Note the cross-walls in the septate hypha.

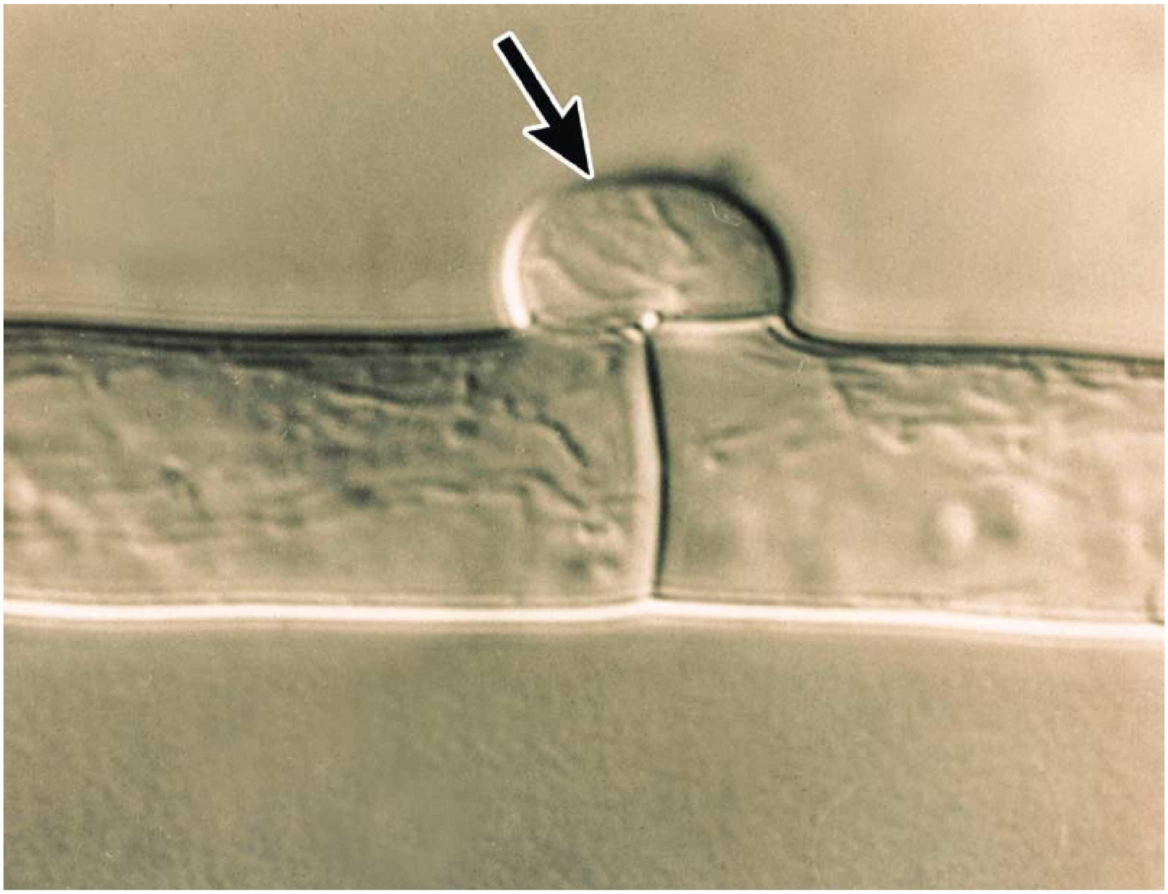


Figure 2.5.
Hypha with a septum and a clamp connection (arrow).

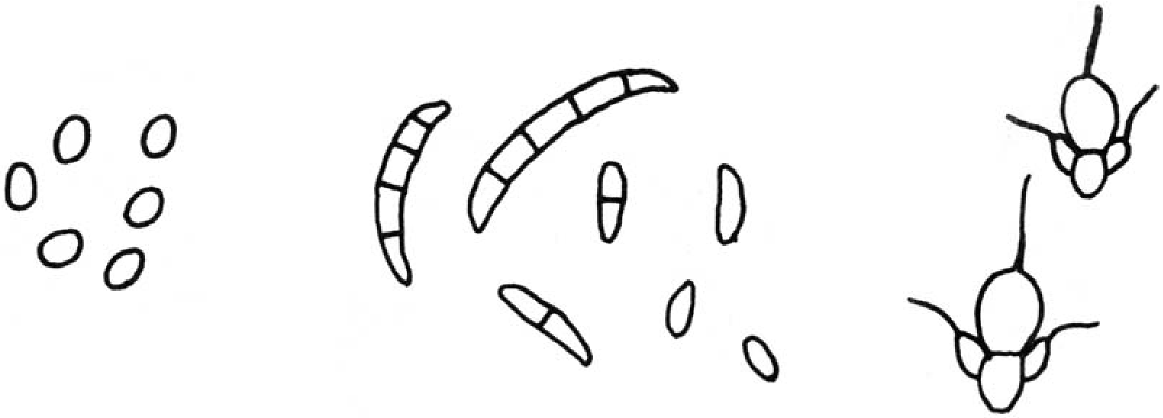


Figure 2.6.
Fungal spores of various shapes.

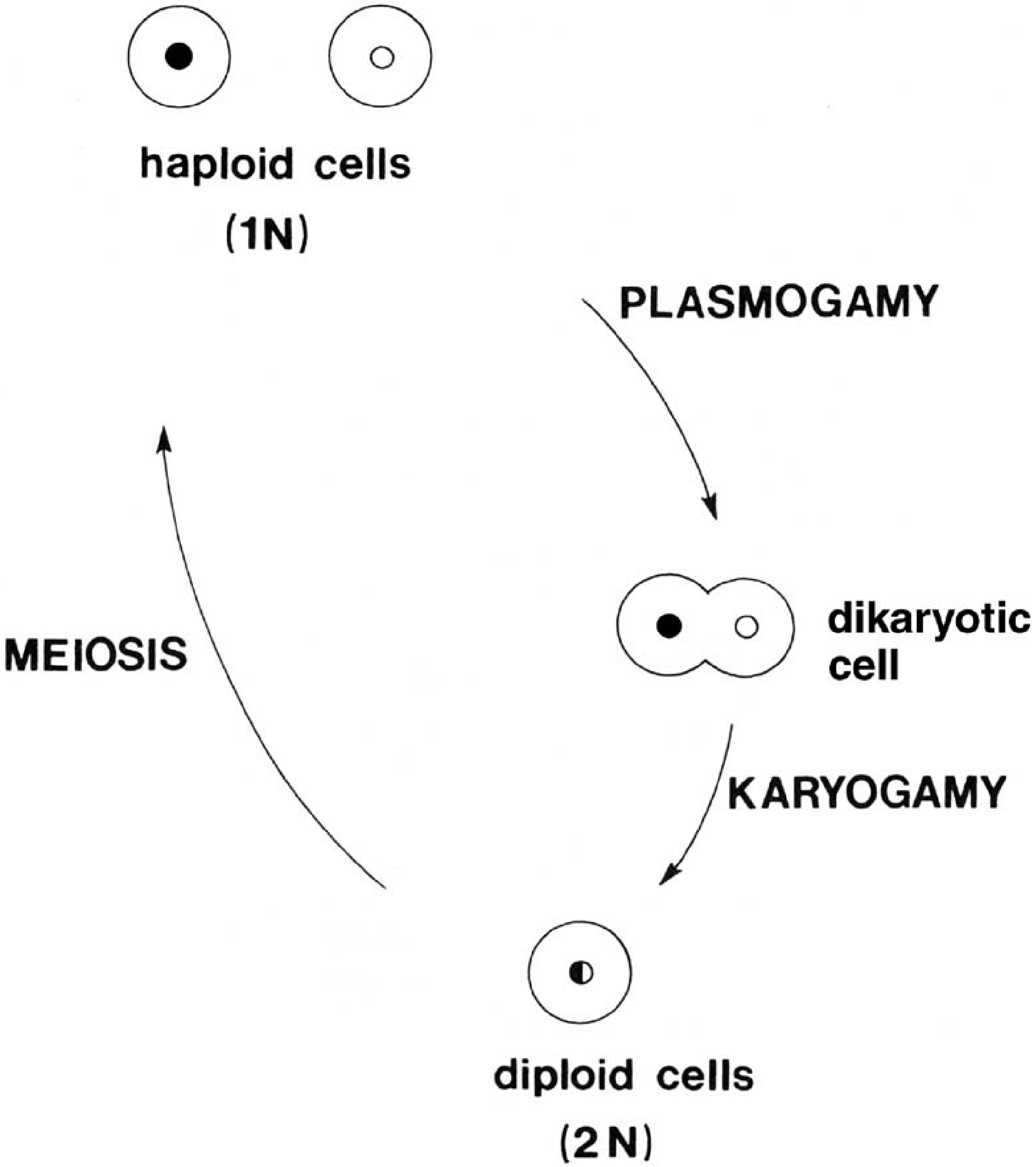

haploid cells
(1N)
PLASMOGAMY
dikaryotic
cell
KARYOGAMY
diploid cells
(2 N)
MEIOSIS

Figure 2.7.
The sexual cycle.

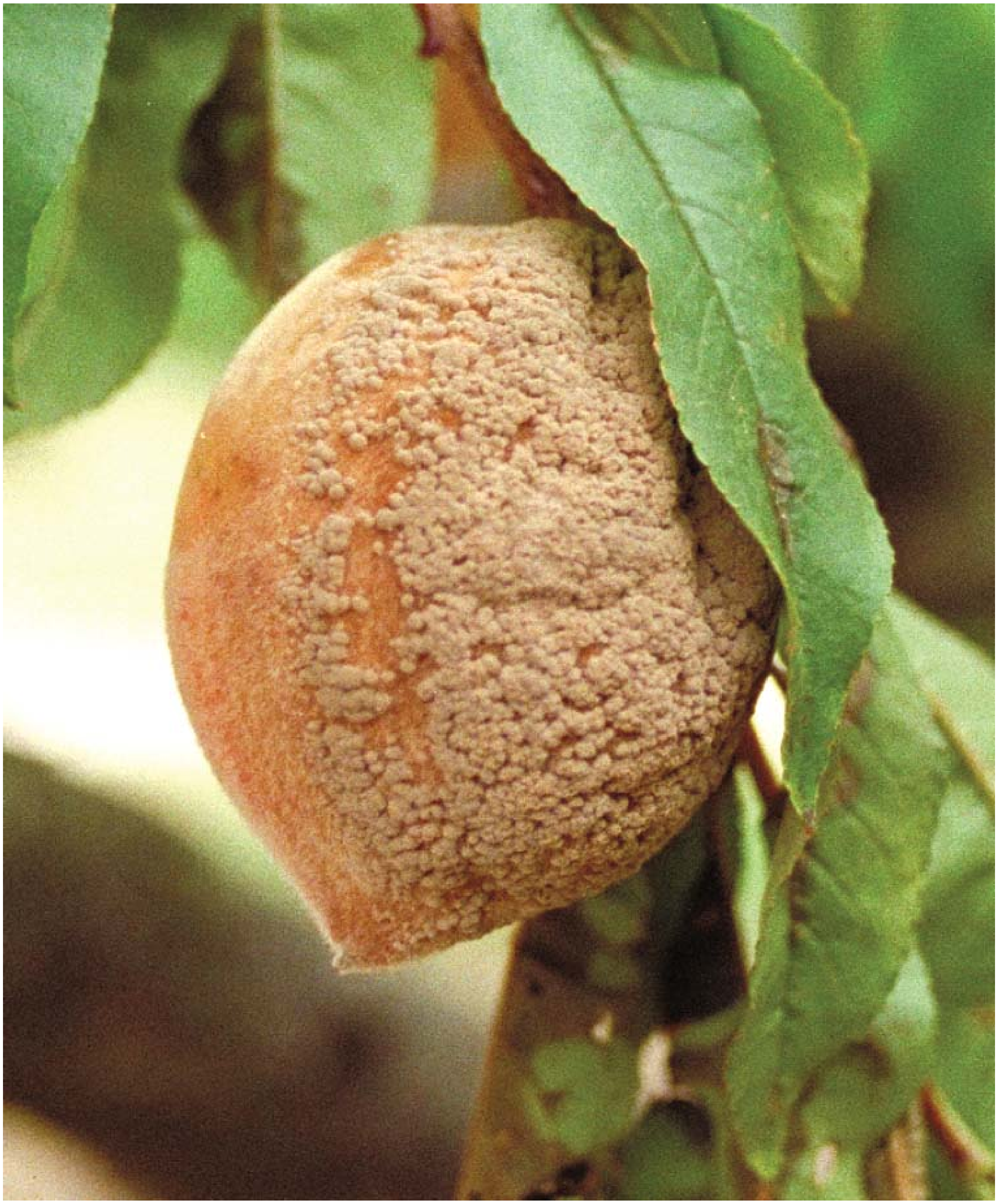


Figure 2.8.
Peach with brown rot. The affected area is covered with spores, a sign of the pathogen.

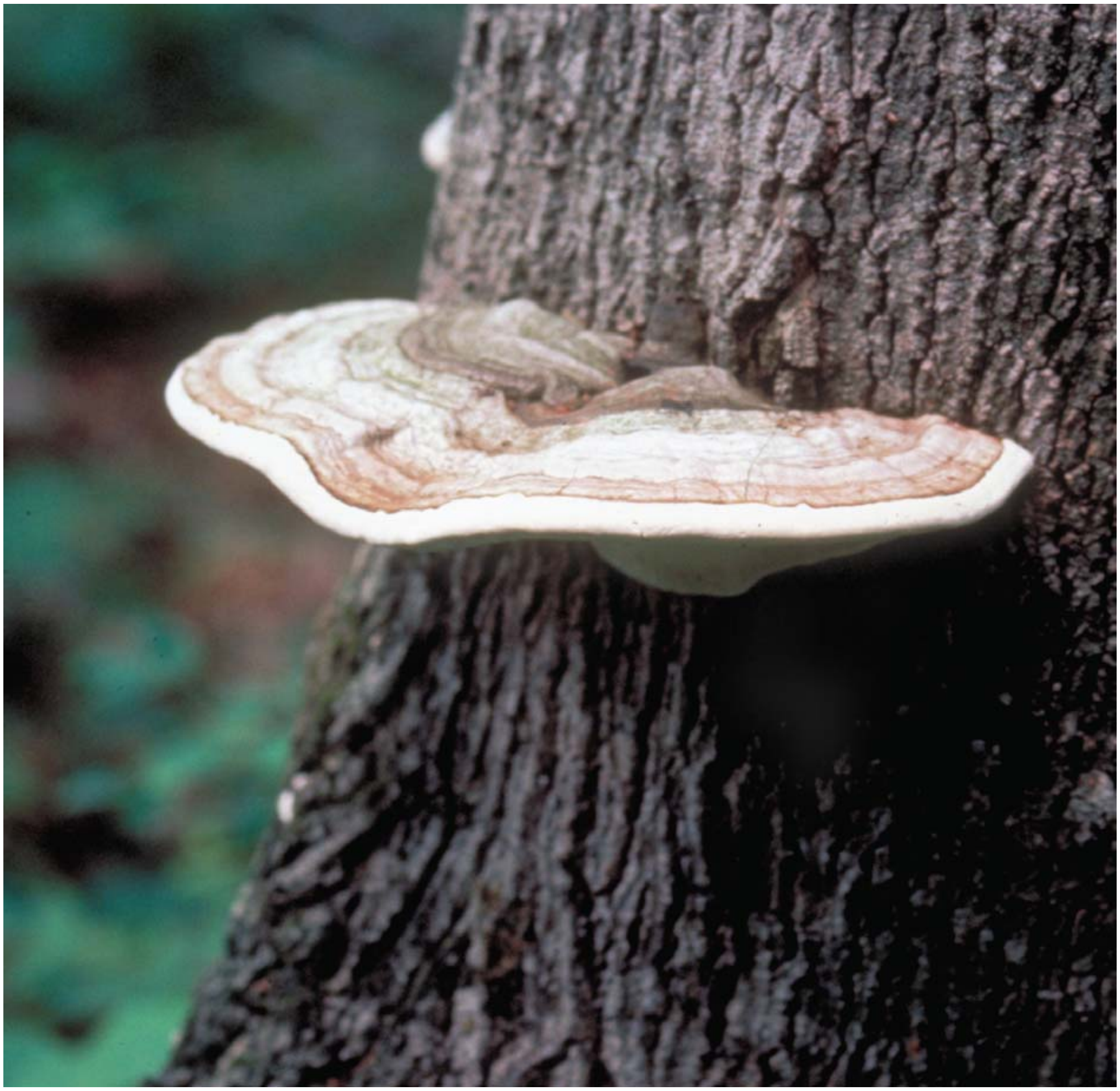


Figure 2.9.
Fruiting body (conk) of a wood decay fungus, a sign of the pathogen.

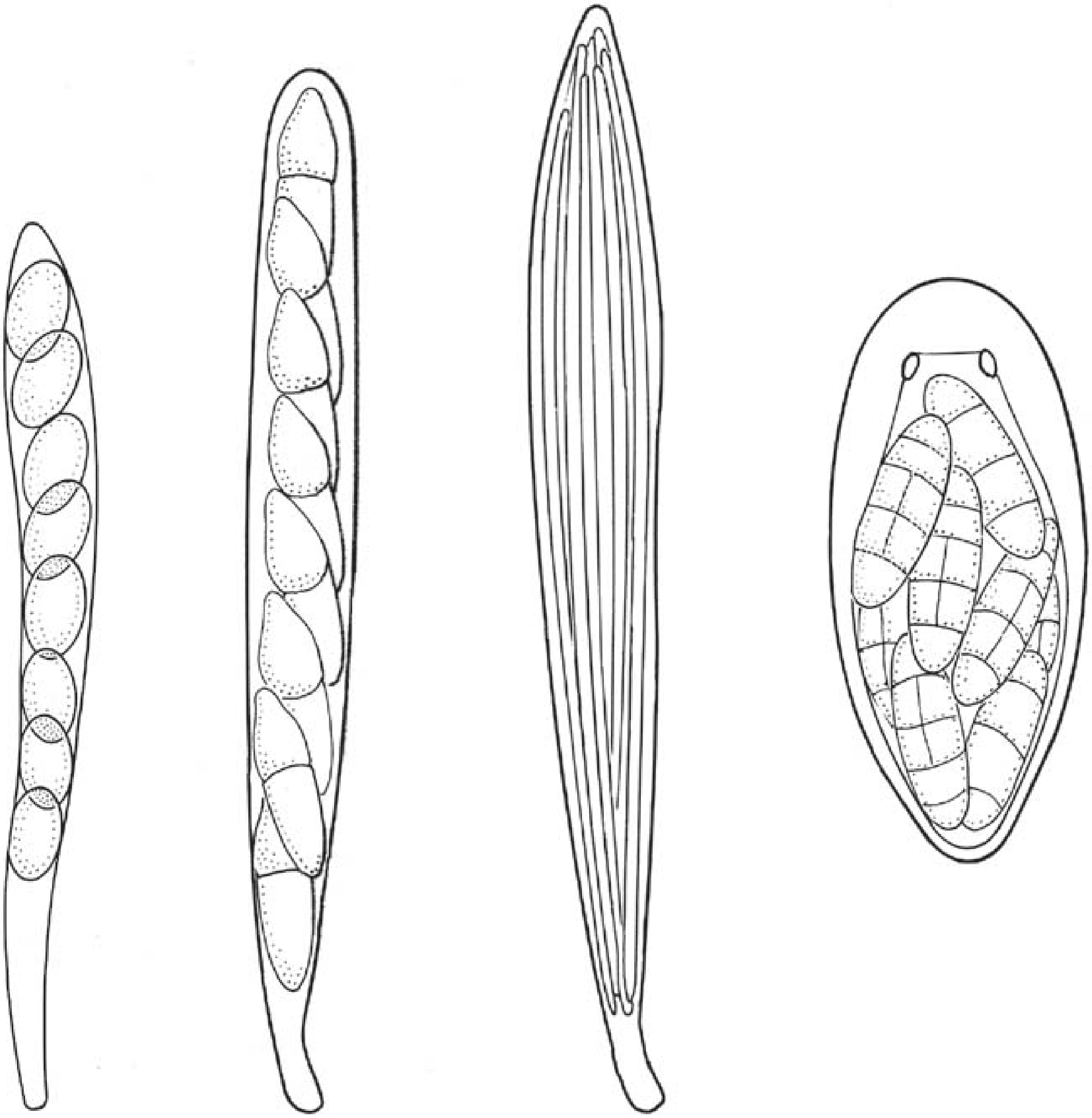


Figure 2.10.
Examples of asci (the outer sac in each diagram) and ascospores (usually eight per ascus). The shape and size of the ascus and ascospores vary greatly.

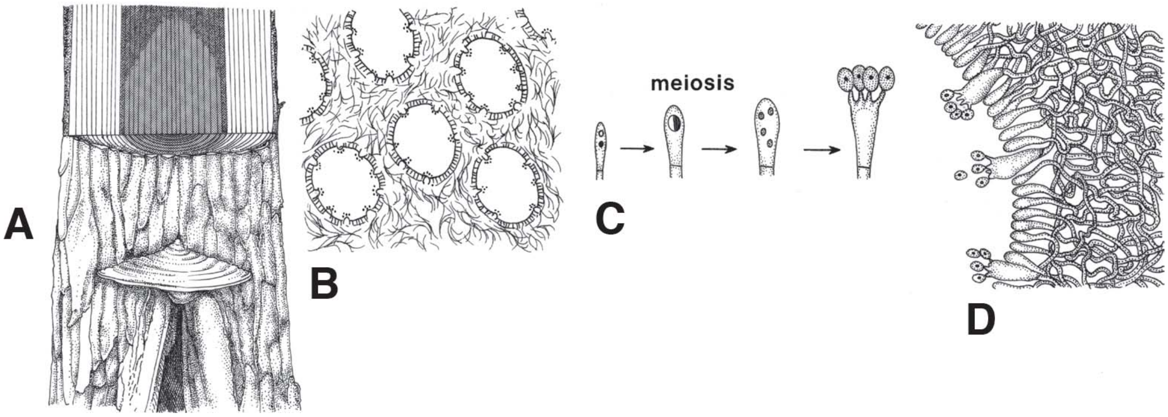


Figure 2.11.
Reproduction of a basidiomycete. **A**, Fruiting body. **B**, The lower surface of a fruiting body, containing numerous pores. **C**, Formation of basidiospores following meiosis. **D**, Basidia and basidiospores lining the pores in a fruiting body.

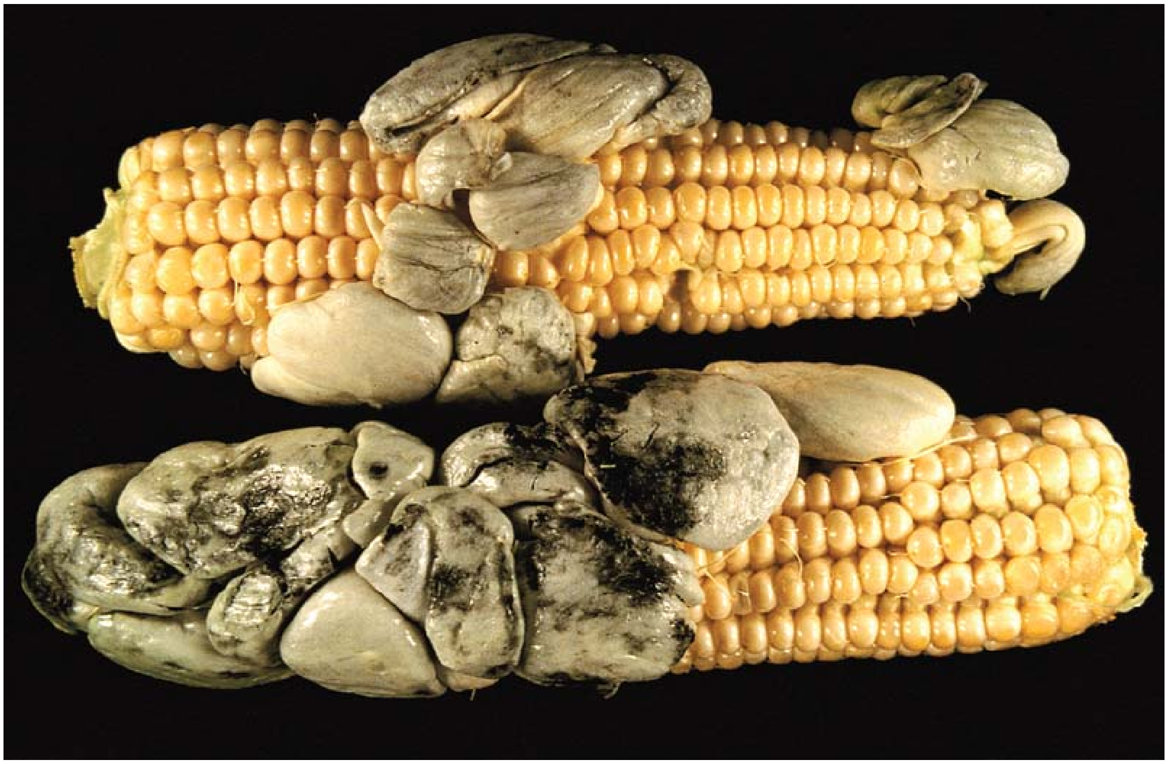


Figure 2.12.
Corn smut, caused by *Ustilago maydis*, a basidiomycete.

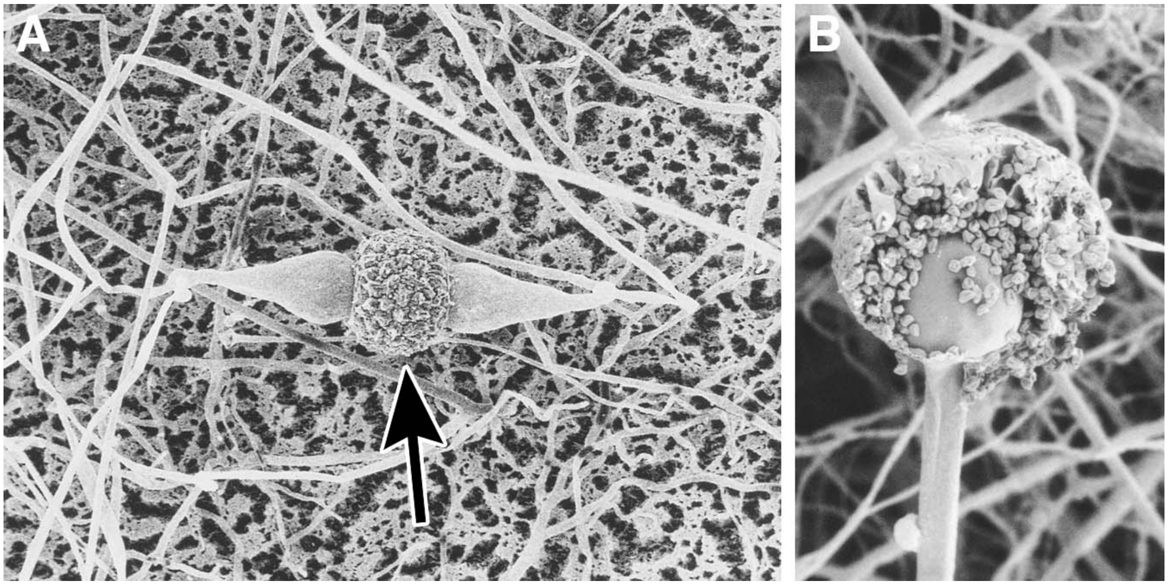


Figure 2.13.
Zygomycete spores. **A**, Zygospore (sexual). **B**, Sporangium and sporangiospores (asexual).

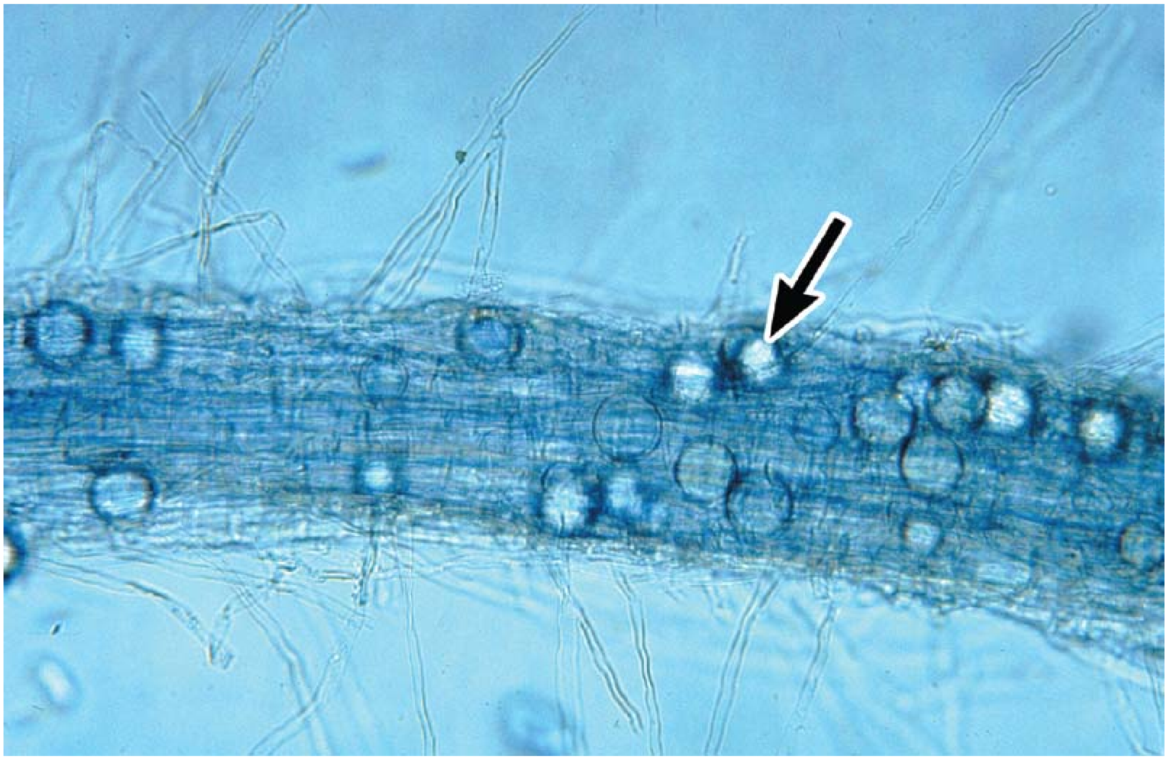


Figure 2.14.
Thick-walled, sexual oospores (arrow) of *Pythium* sp. in a turfgrass root.

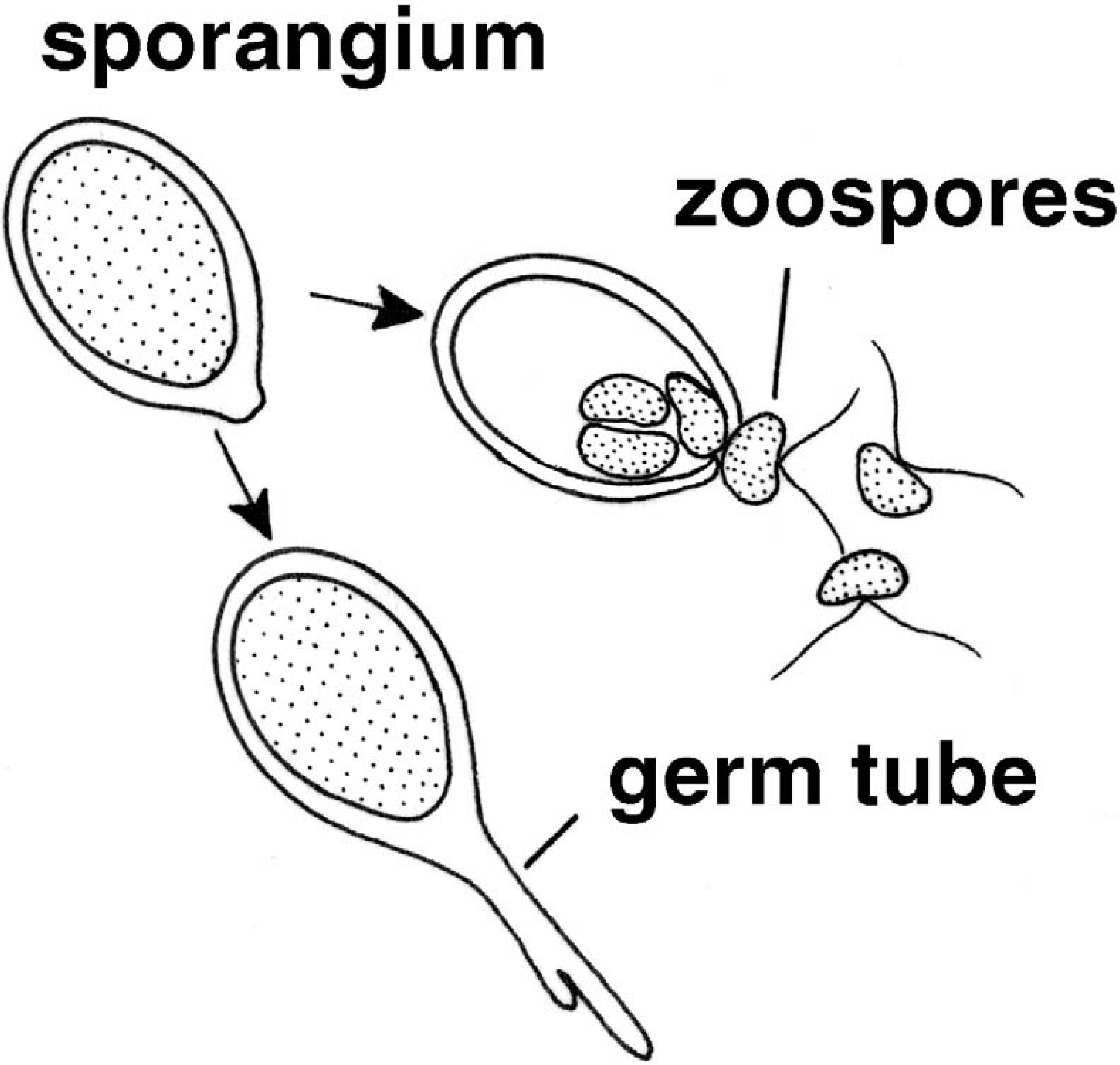

sporangium
zoospores
germ tube

Figure 2.15.
Sporangium of an oomycete producing asexual zoospores (indirect germination). Sometimes a sporangium functions as a single spore and produces a germ tube (direct germination).

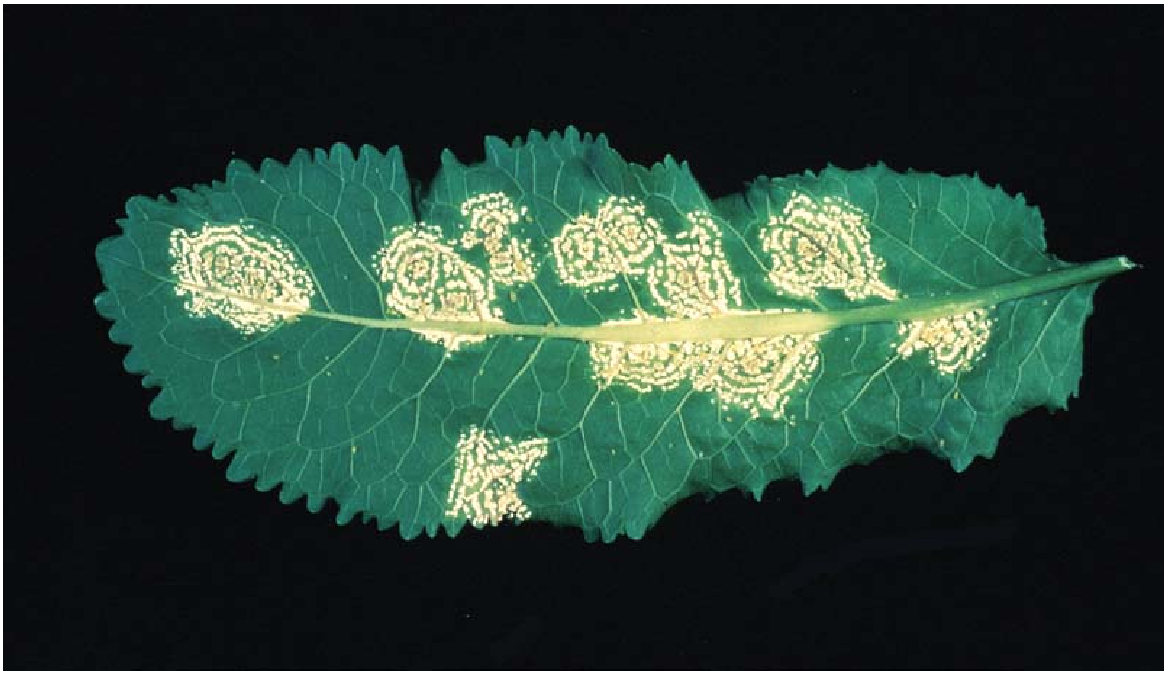


Figure 2.16.
White rust (*Albugo candida*) on a brassica (crucifer) leaf.

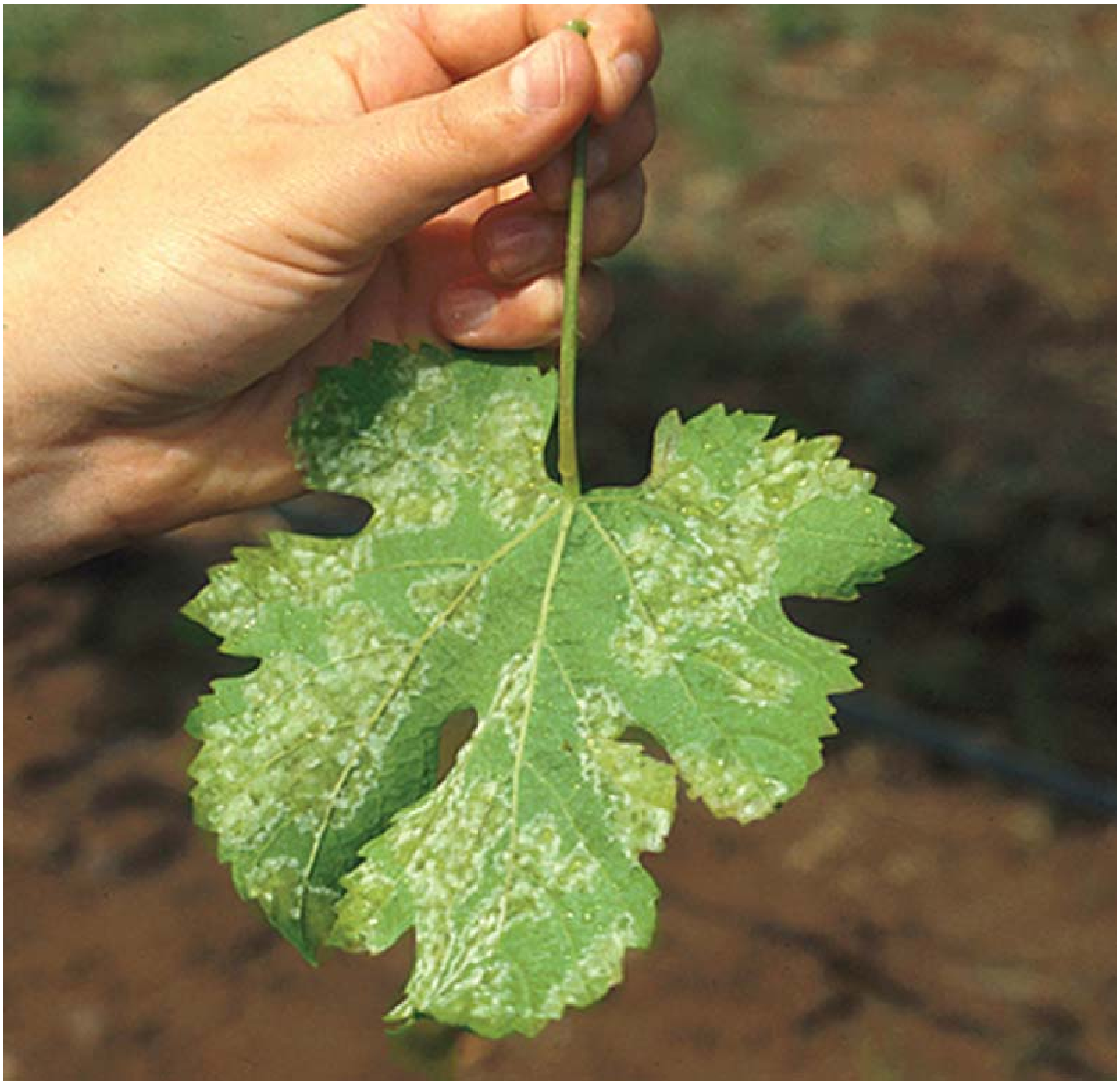


Figure 2.17.
Downy mildew on the lower surface of a grape leaf. Note the sporangia and sporangiophores.

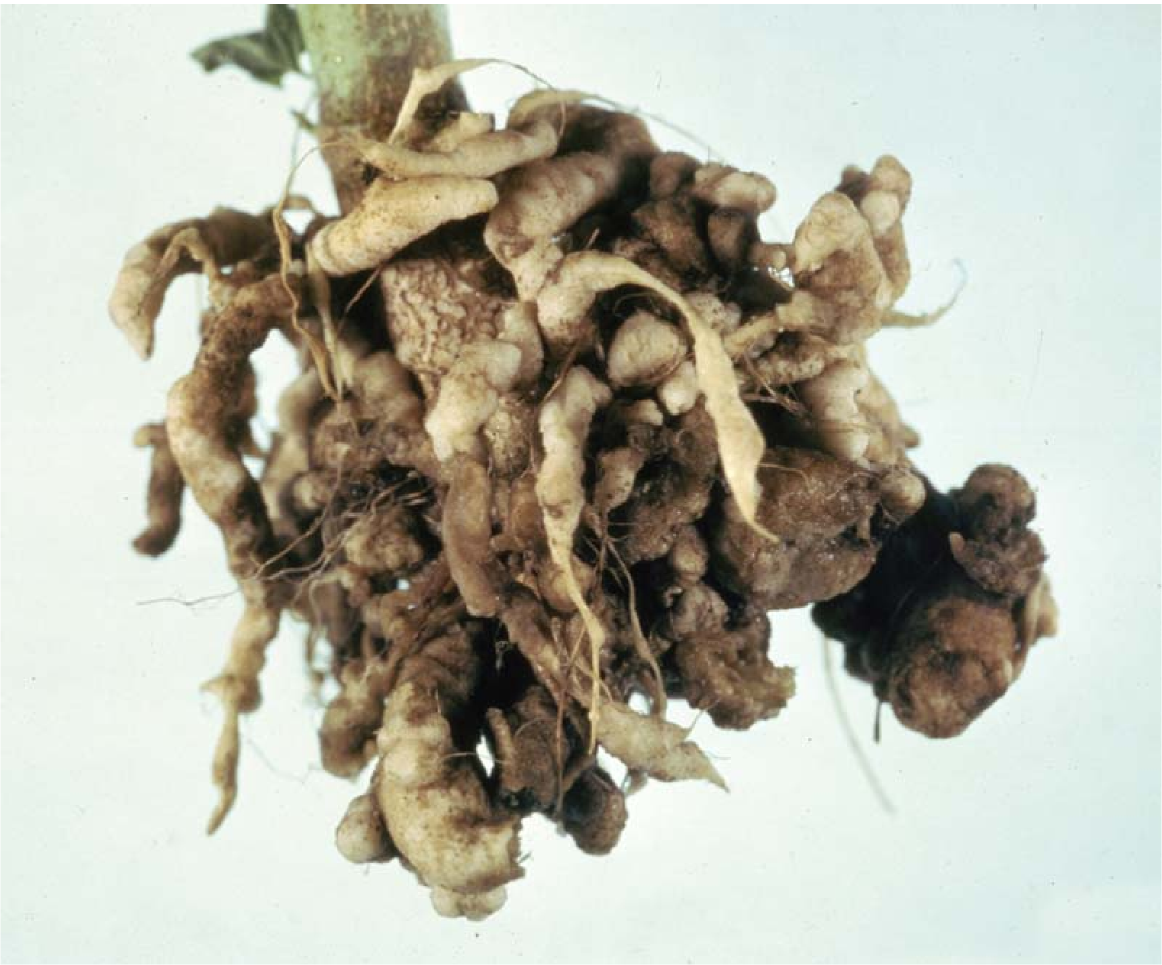


Figure 2.18.
Clubroot of broccoli, caused by *Plasmodiophora brassicae.*

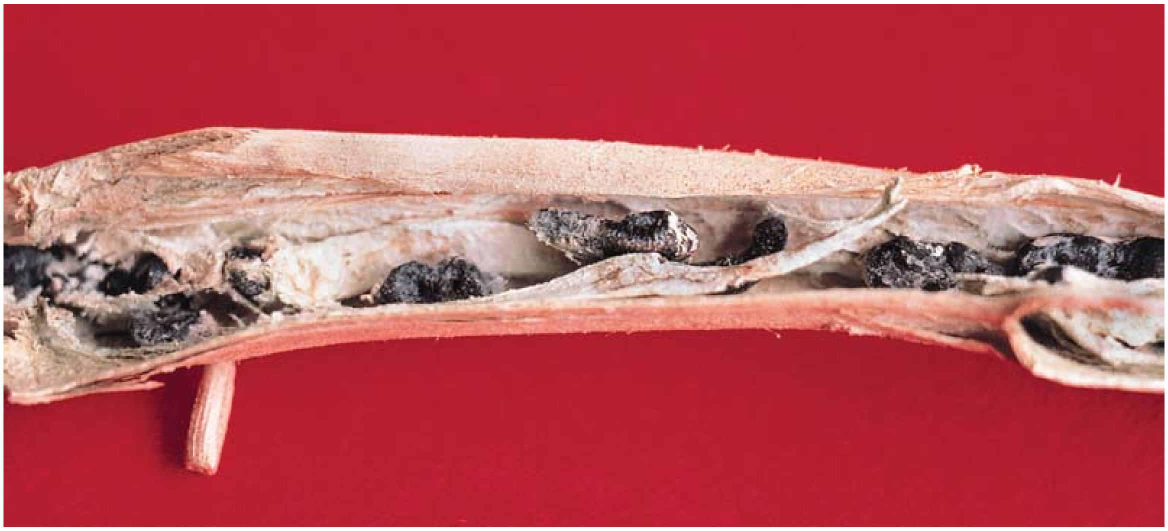


Figure 2.19.
Large, black sclerotia of the white mold fungus (*Sclerotinia sclerotiorum*) in a tomato stem.

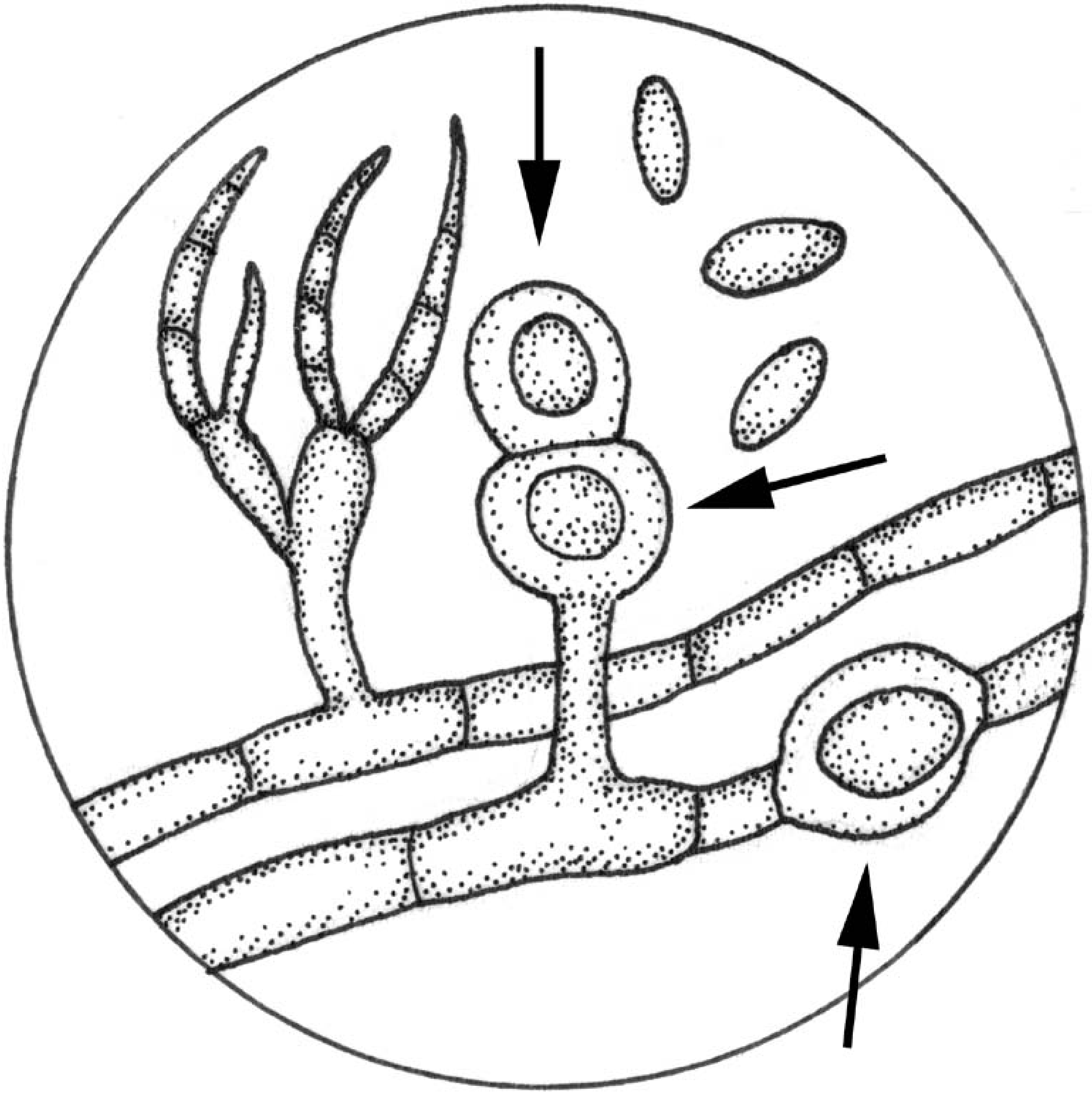


Figure 2.20.
Thick-walled chlamydospores of *Fusarium* sp. (arrows).

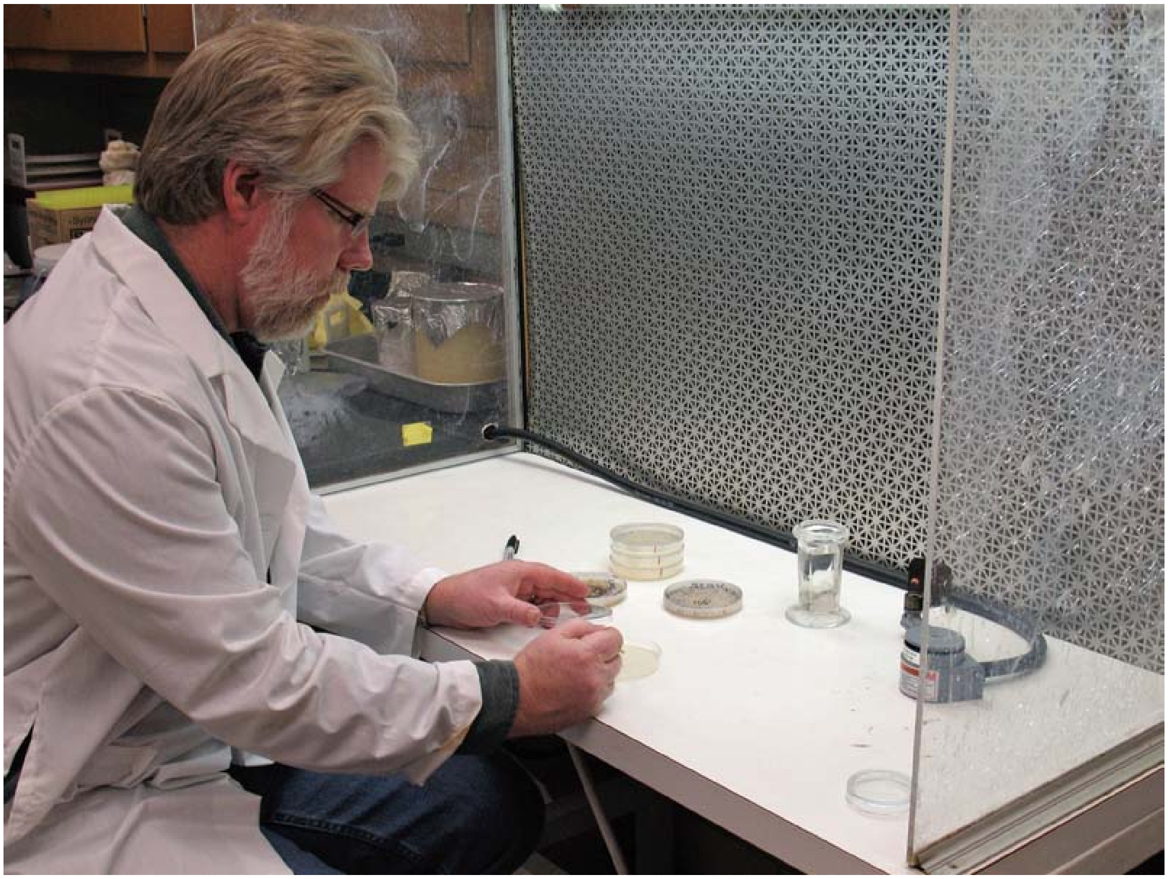


Figure 2.21.
Plant pathologist working in a laminar flow hood and using sterile tools to isolate and transfer fungi in petri dishes.

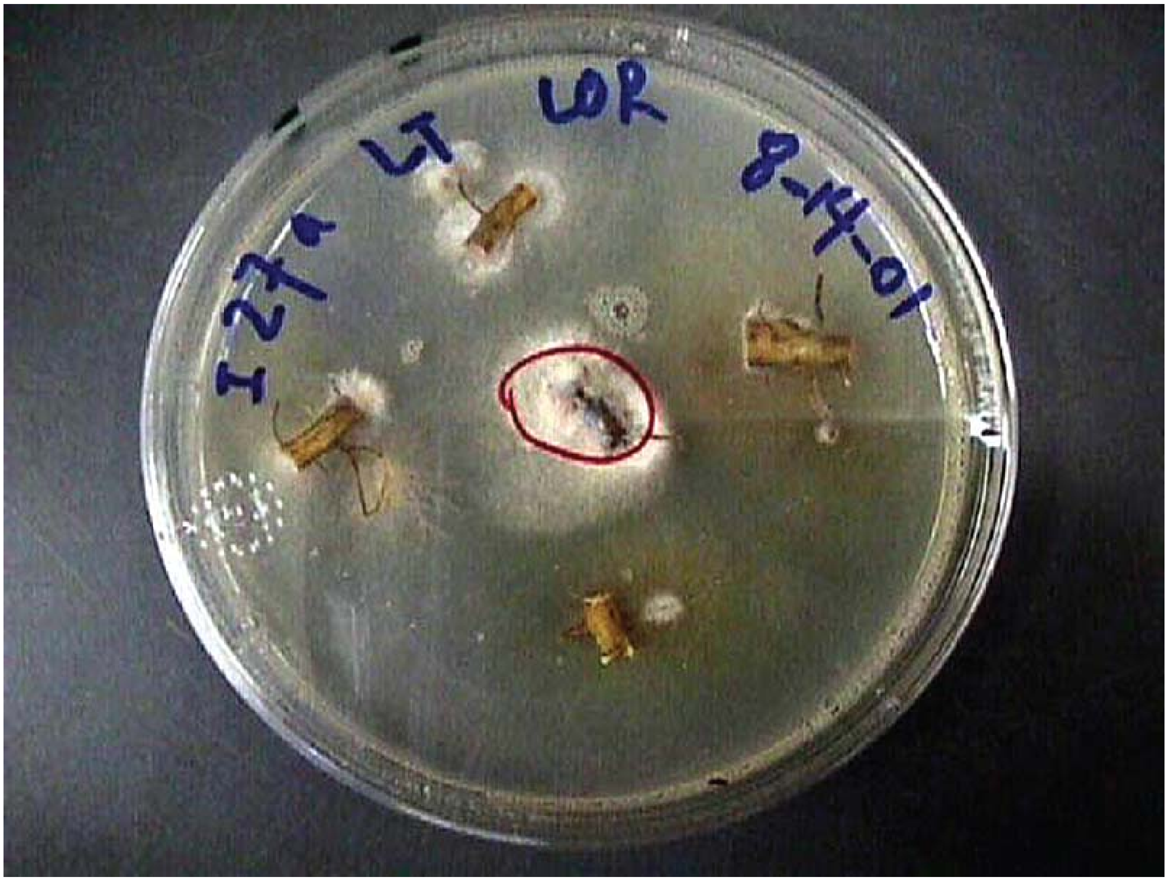

I 27a LT LOR 8-14-01

Figure 2.22.
Isolation of a fungal pathogen from plant tissue. Note variable growth.

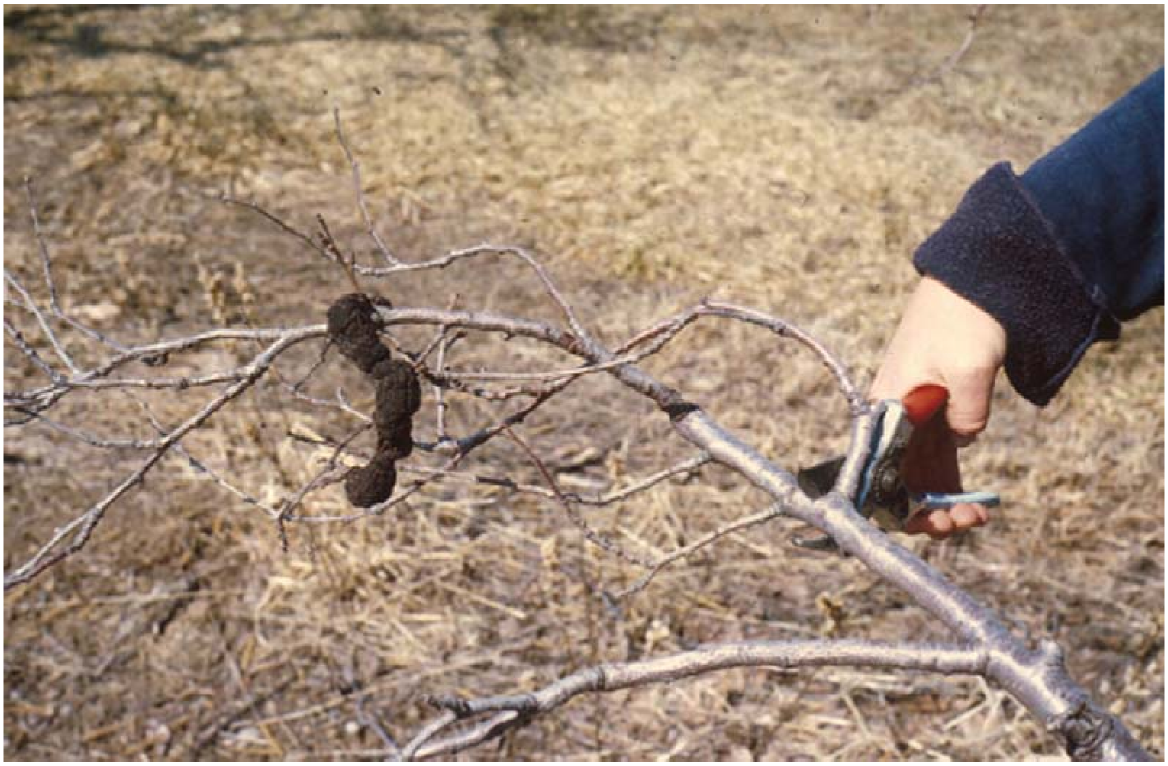


Figure 2.23.
Eradication: pruning black knot cankers from a cherry tree.

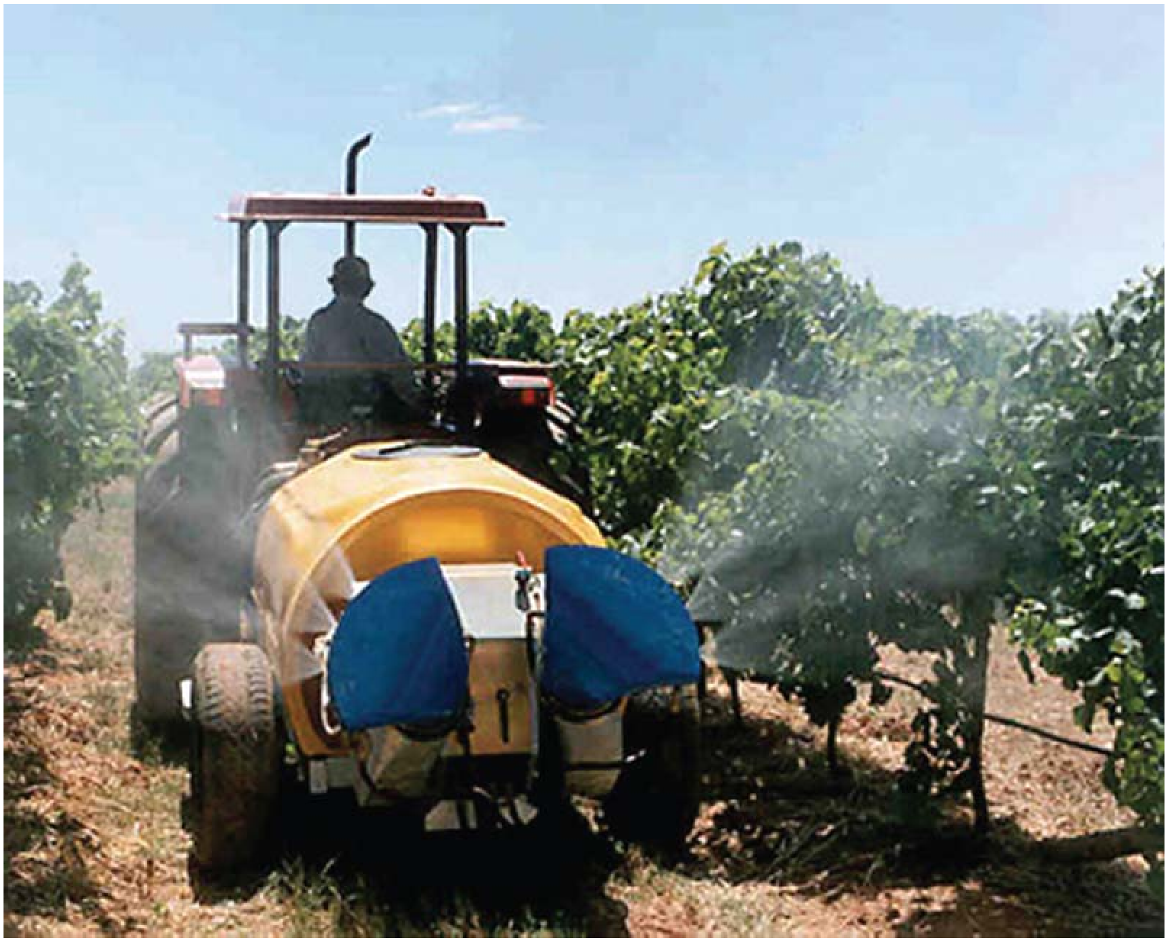


Figure 2.24.

Protection: fungicide application in a vineyard.

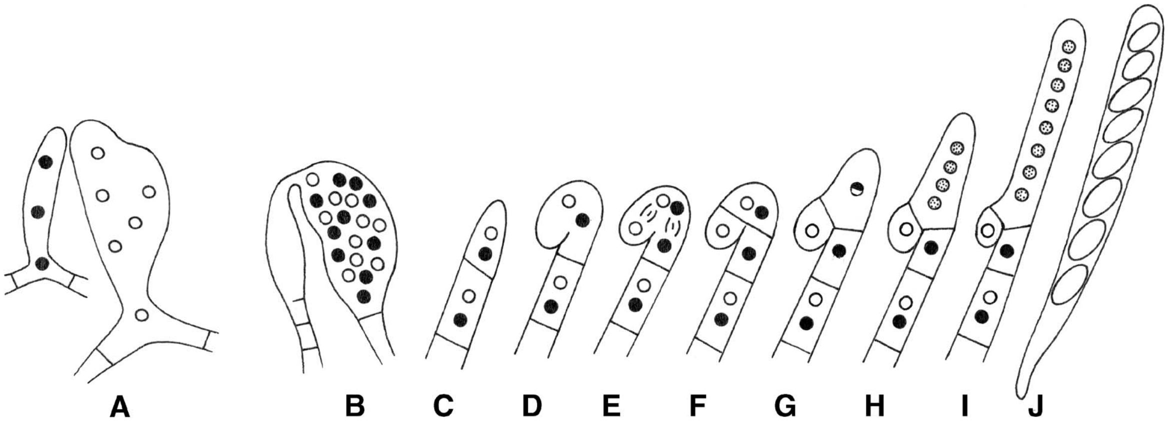


Figure 2.25.

Generalized process of ascus formation. **A**, Male structure (often an antheridium) and female structure (ascogonium). **B**, Plasmogamy: the male structure contributes nuclei to the female structure. **C**, A limited dikaryotic mycelium develops. **D-F**, Through a complex mechanism, the ascus mother cell forms. **G**, Karyogamy: the two nuclei fuse to form a single diploid nucleus. **H**, Meiosis produces four haploid nuclei. **I**, In many ascomycetes, mitosis produces eight haploid nuclei and the ascus elongates. **J**, A spore wall forms around each nucleus to produce eight ascospores.

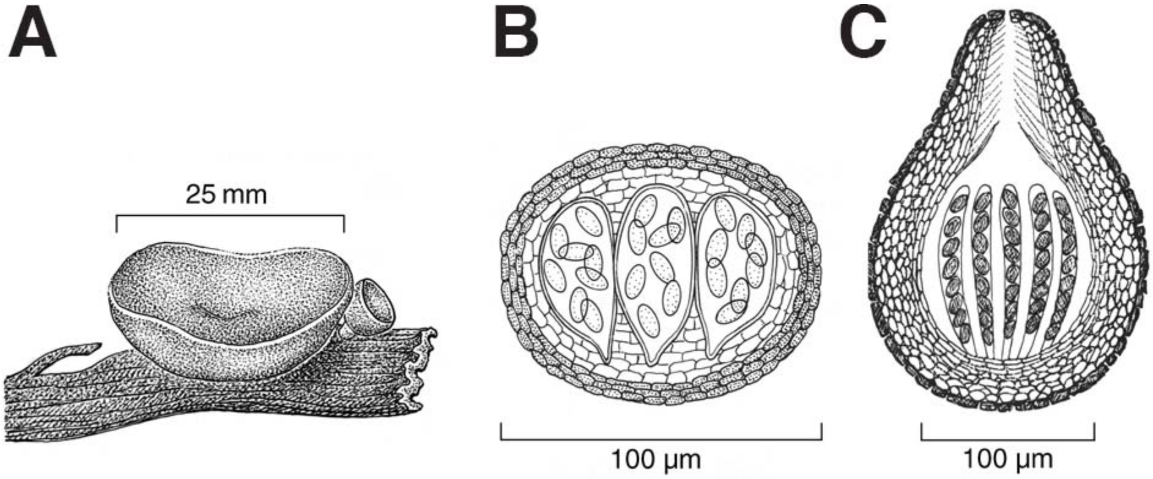

A
B
C
25 mm
100 µm
100 µm

Figure 2.26.
Ascomata with unitunicate asci. **A**, Apothecium. **B**, Chasmothecium (cleistothecium). **C**, Perithecium. Note the asci and ascospores.

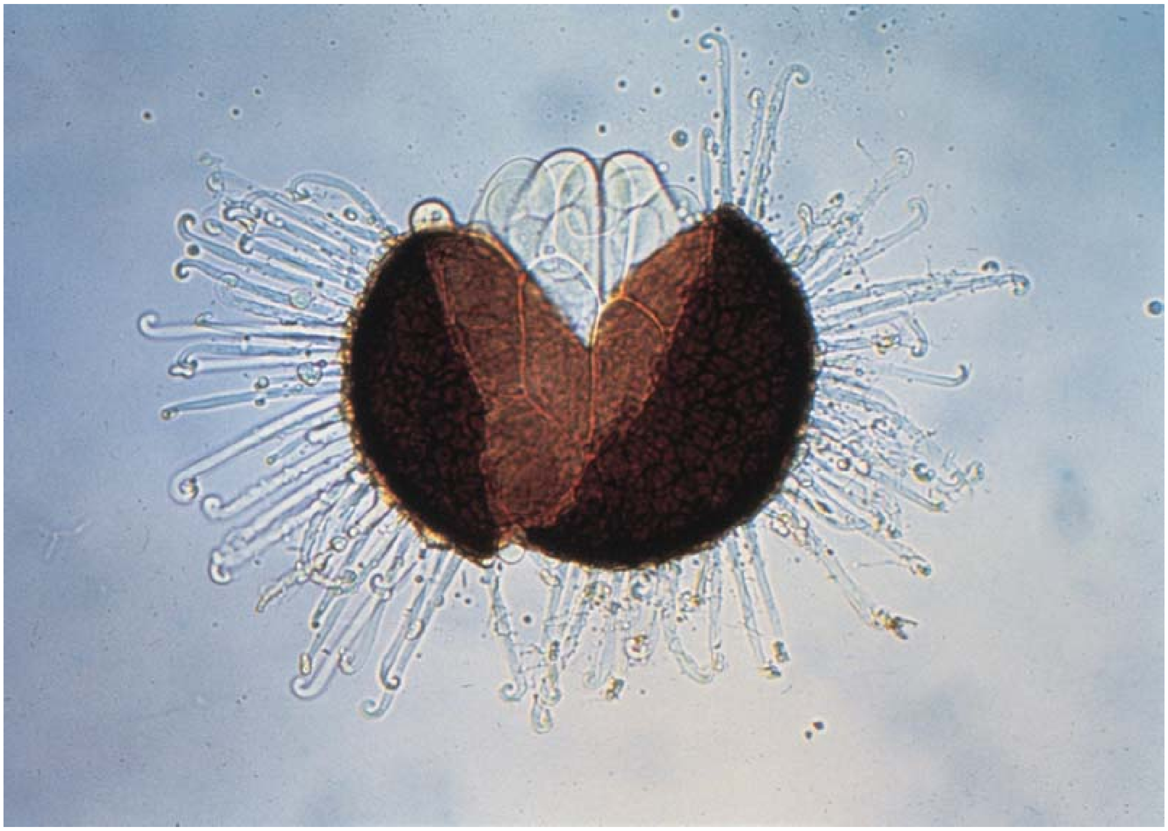


Figure 2.27.
Chasmothecium (cleistothecium) of a powdery mildew fungus opening to reveal asci and ascospores.

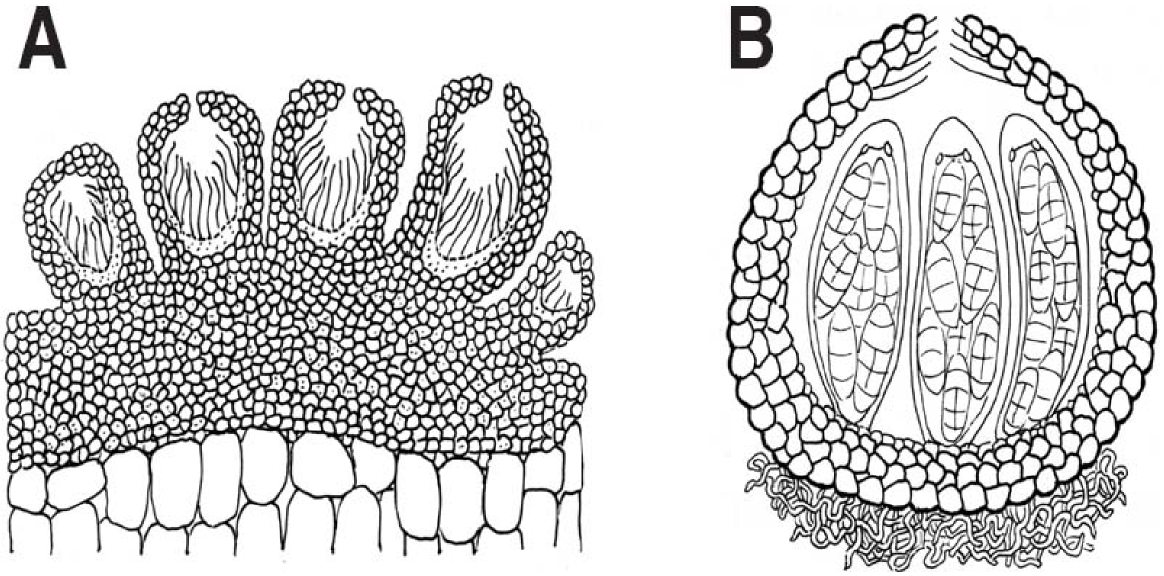
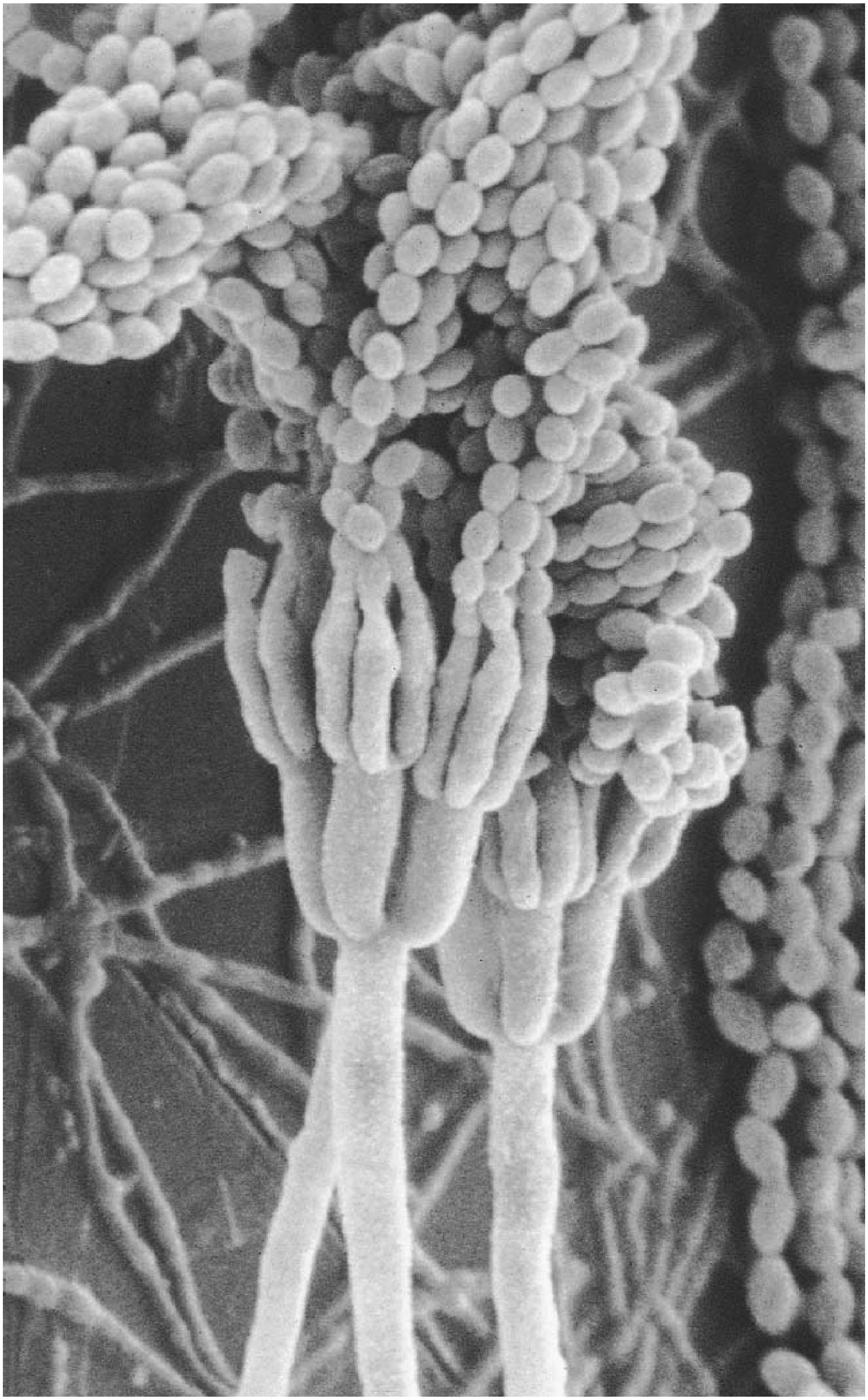


Figure 2.28.
Ascomata with bitunicate asci. **A**, Ascostroma with multiple locules filled with asci. **B**, Pseudothecium. Note the double wall of the bitunicate asci.



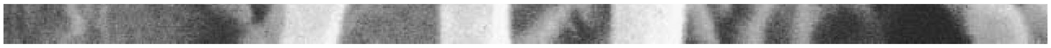


Figure 2.29.
Conidia on a conidiophore (*Penicillium* sp.).

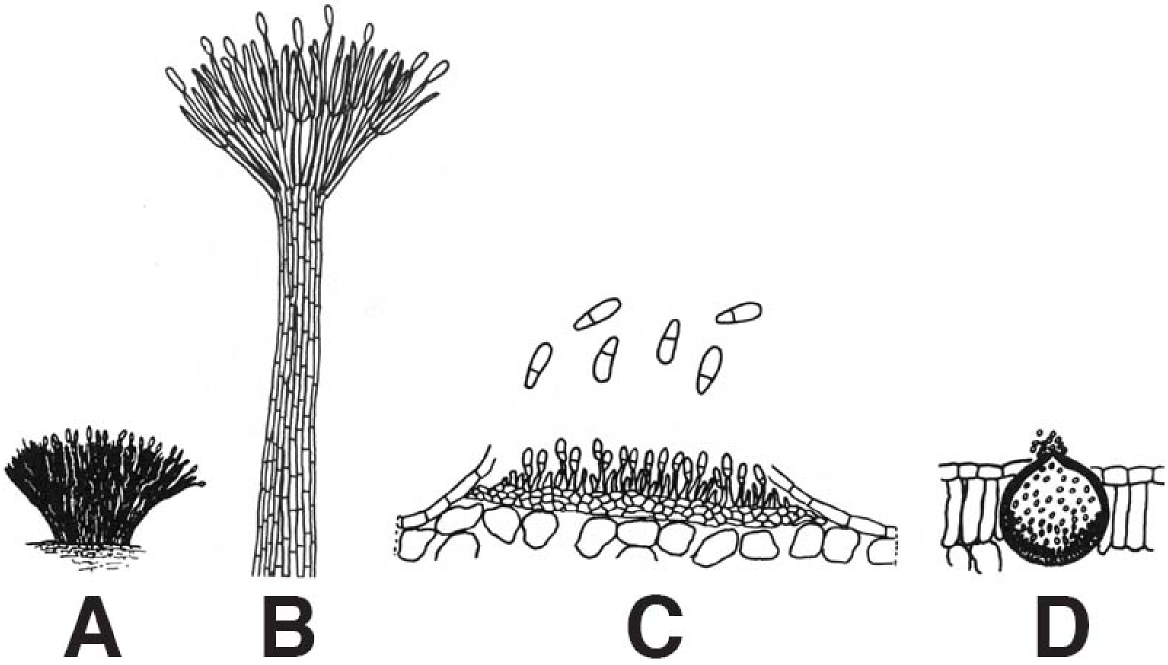
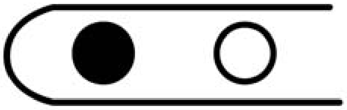
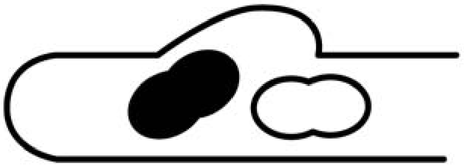
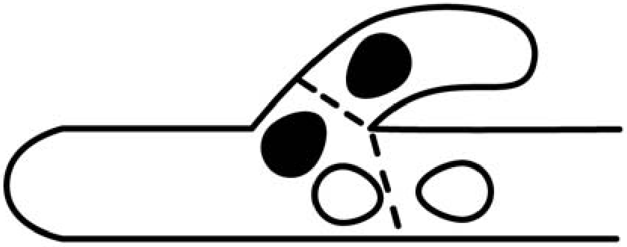
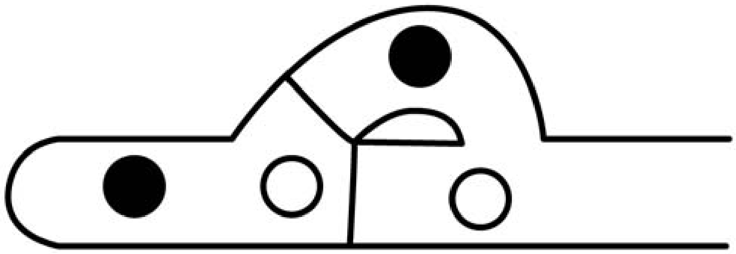


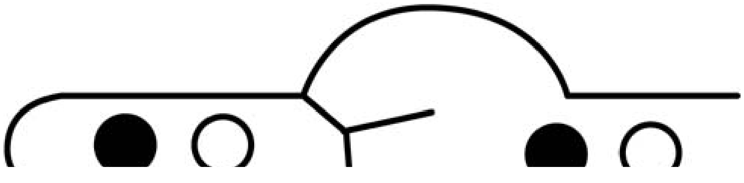
Figure 2.30.
Conidiomata. **A**, Sporodochium. **B**, Synnema. **C**, Acervulus. **D**, Pycnidium. Note that the conidia are not in asci.











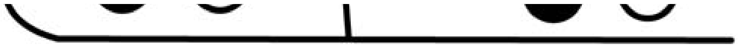


Figure 2.31.
Clamp connection and dikaryotic hypha formed during mitosis.

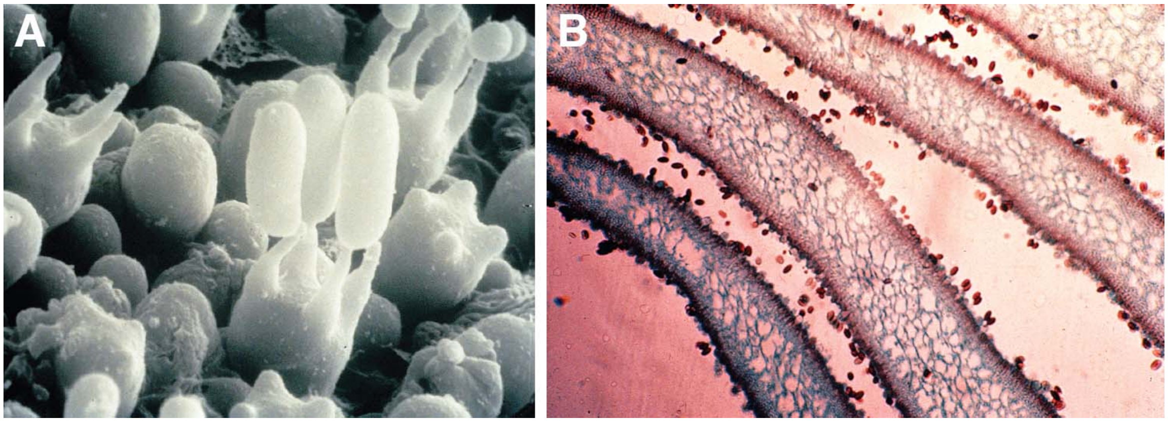


Figure 2.32.
A, Four basidiospores on a basidium. **B**, Basidia and basidiospores along the gills on the lower surface of a mushroom.

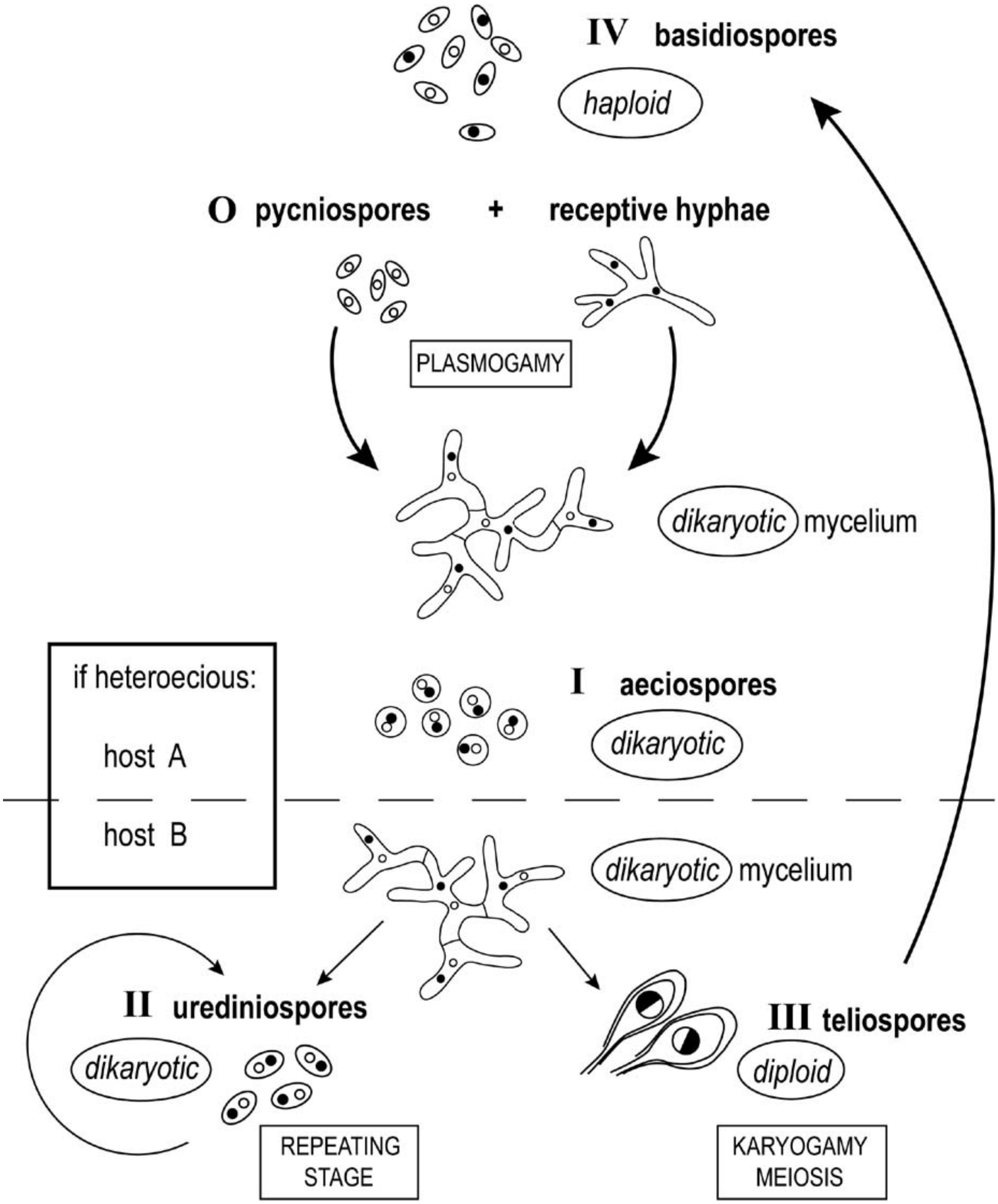

IV basidiospores
haploid
O pycniospores + receptive hyphae
PLASMOGAMY
dikaryotic mycelium
if heteroecious:
host A
host B
I aeciospores
dikaryotic
dikaryotic mycelium
II urediniospores
dikaryotic
REPEATING STAGE
III teliospores
diploid
KARYOGAMY MEIOSIS

Figure 2.33.
Heteroecious, macrocyclic rust life cycle with Roman numerals.

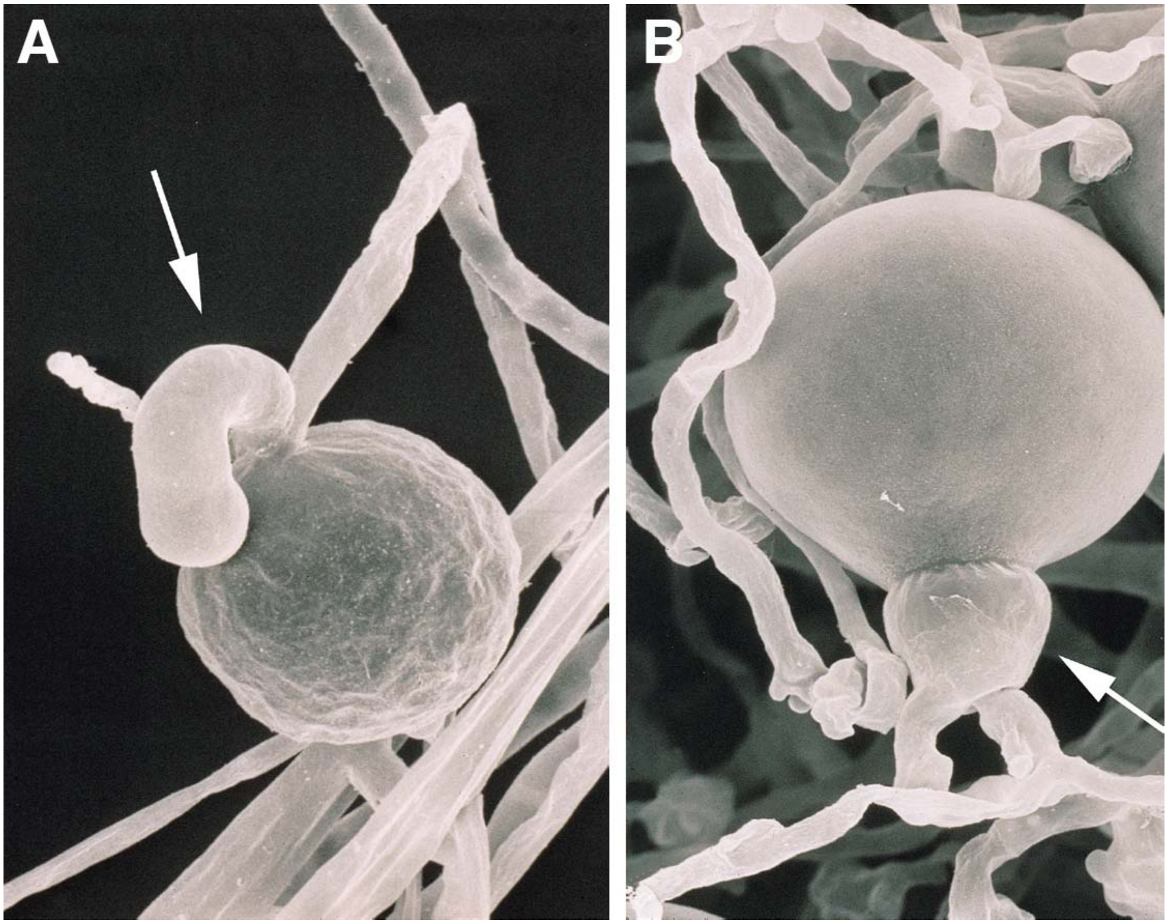


Figure 2.34.
A, Paragynous antheridium (arrow) of *Pythium* sp. **B**, Amphigynous antheridium (arrow) of *Phytophthora* sp.

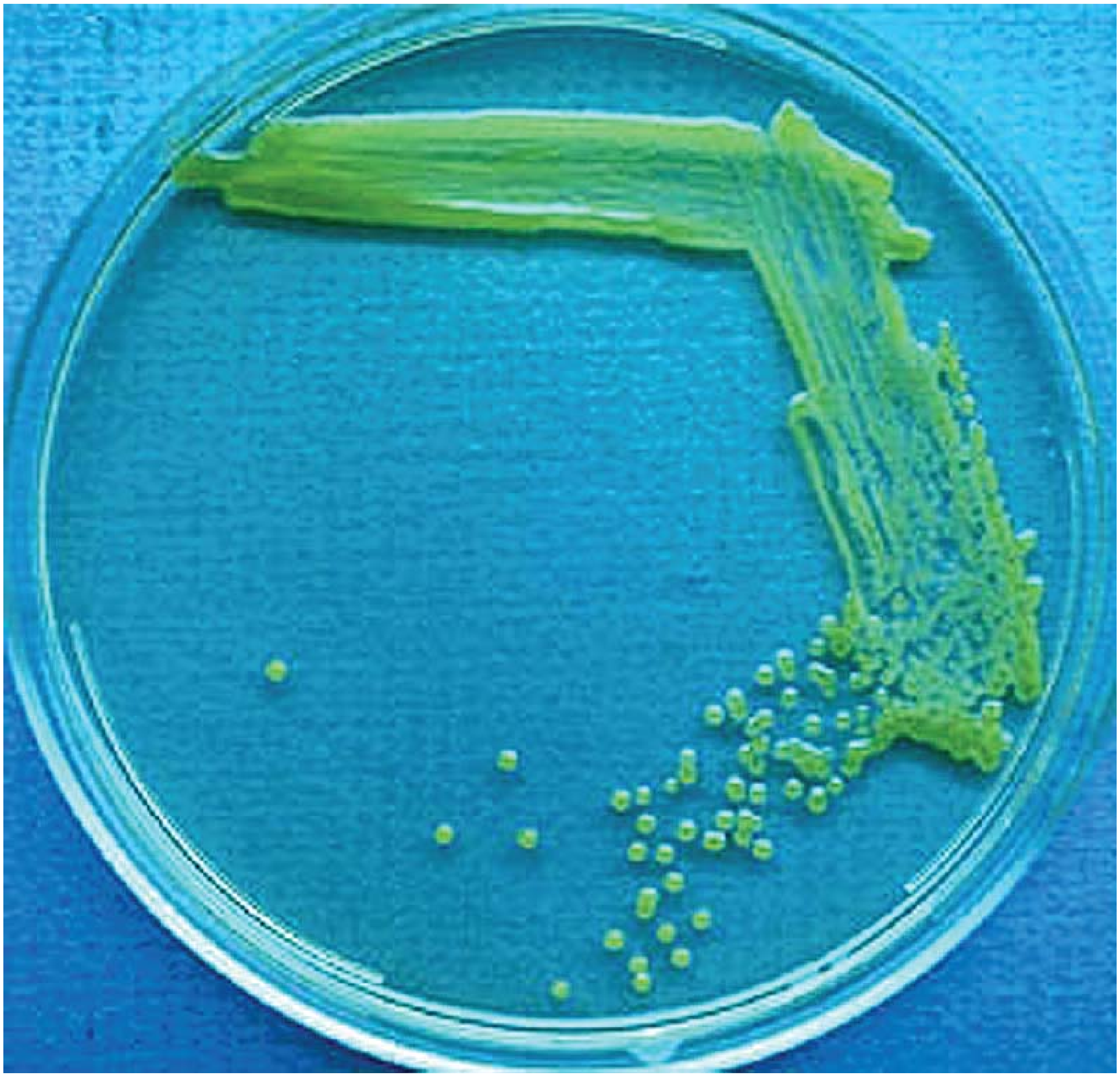


Figure 3.1.
Bacterial colonies on a nutrient medium. Note that the bacteria have been diluted through streaking. The small, round colonies are made up of thousands to millions of cells, and each probably grew from a single bacterium.

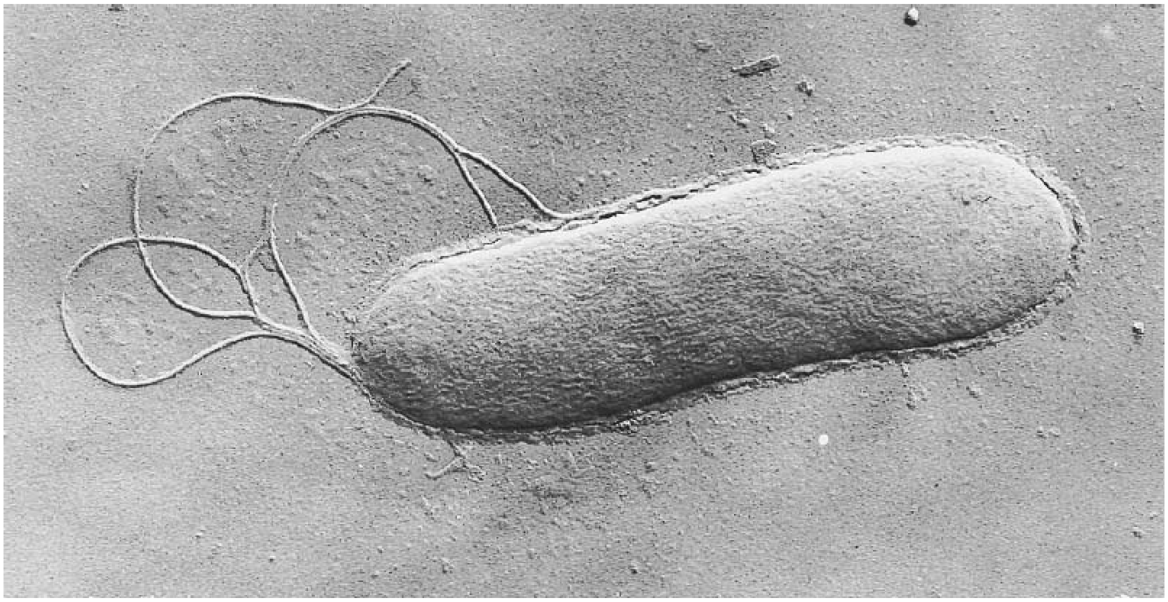


Figure 3.2.
A bacilliform bacterium, typical of plant pathogens. Note that it has polar flagella.

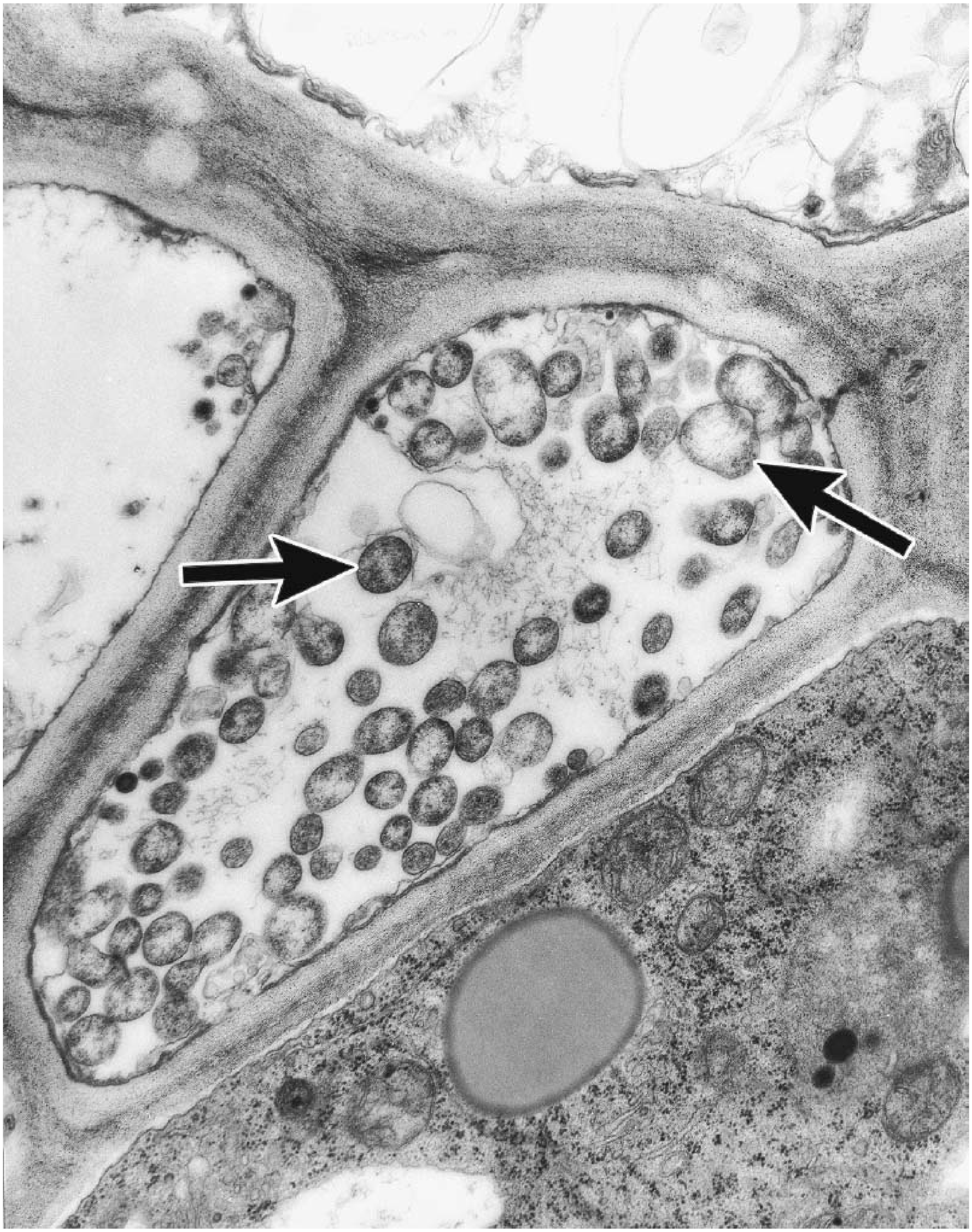


Figure 3.3.

Phytoplasmas (arrows) in a phloem cell of an infected plant. Note that the phytoplasmas are pleomorphic (variably shaped).

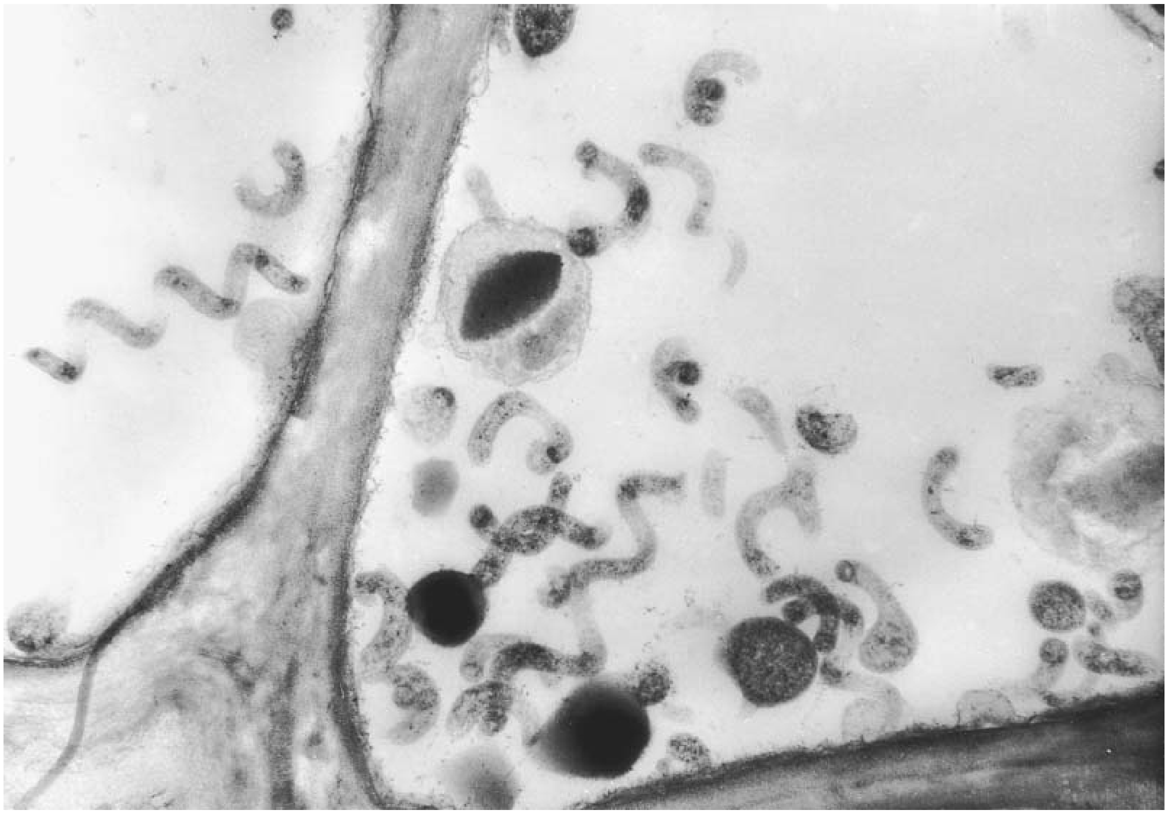


Figure 3.4.
Spiroplasmas in phloem cells of an infected corn plant. Note the helical shape.

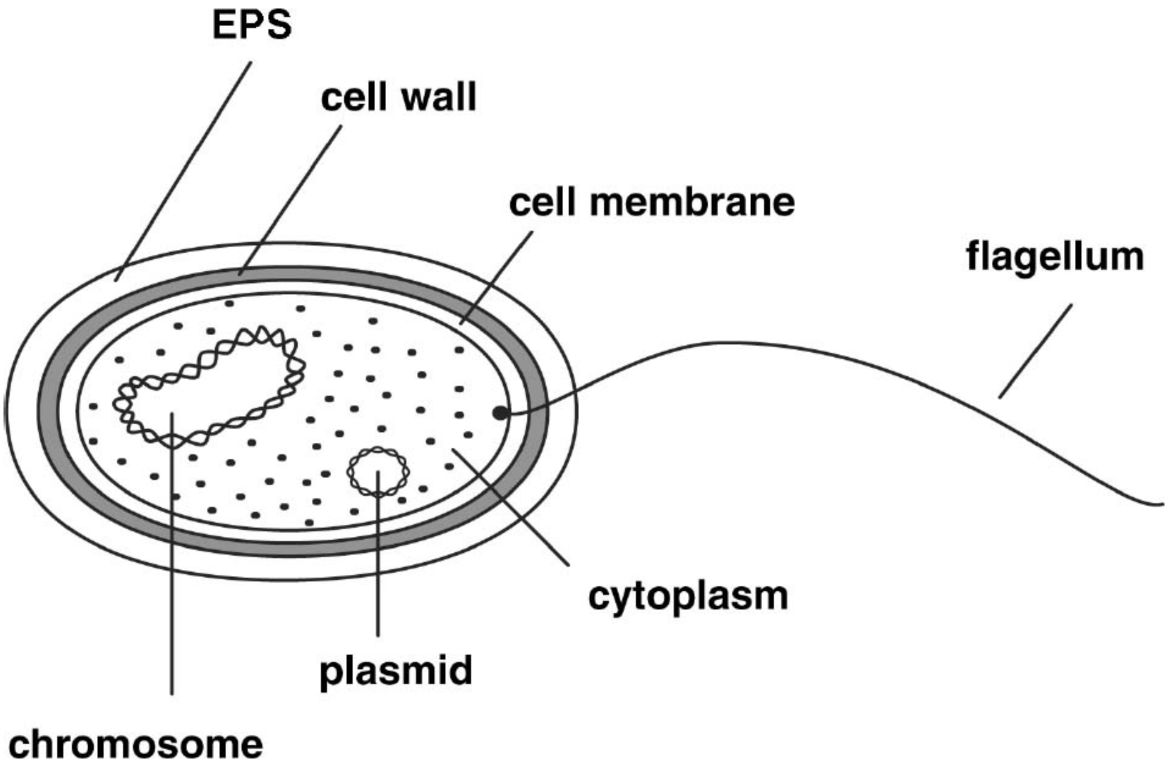

EPS
cell wall
cell membrane
flagellum
cytoplasm
plasmid
chromosome

Figure 3.5.
Bacterial cell. Note the EPS (extracellular polysaccharide) and polar flagellum.

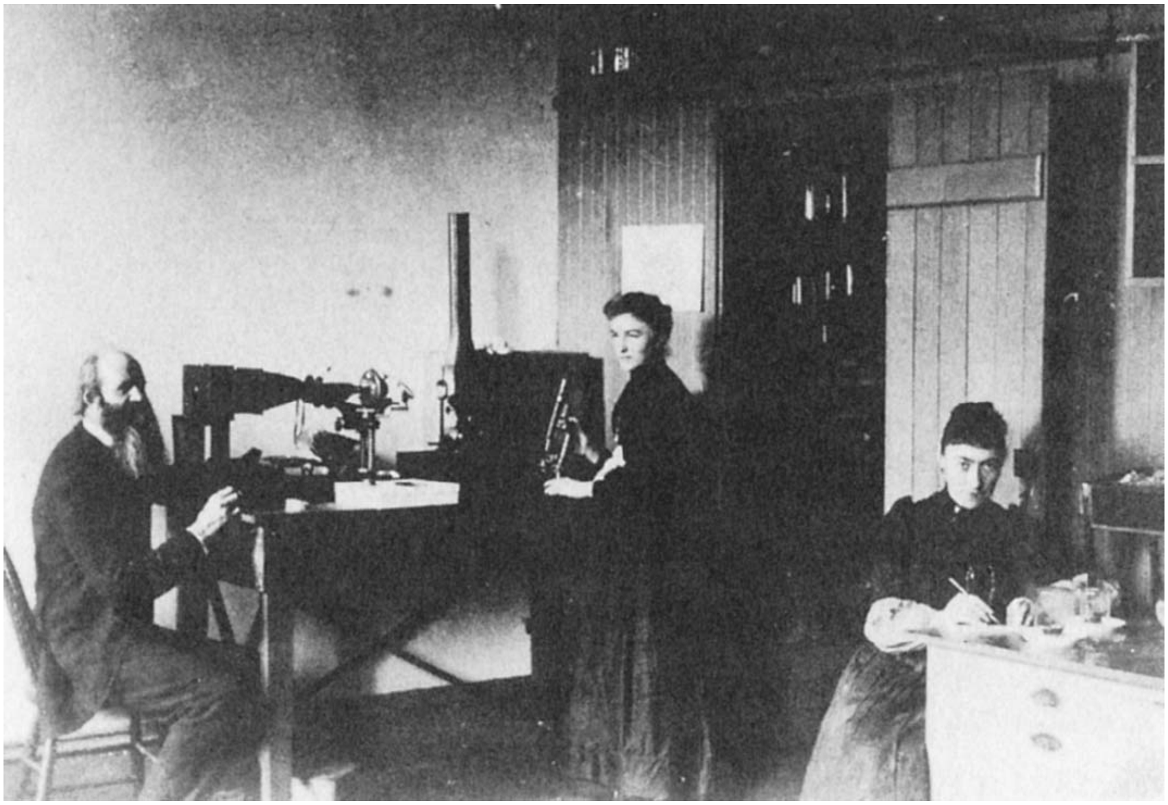


Figure 3.6.
Thomas J. Burrill in a laboratory with students, 1882.

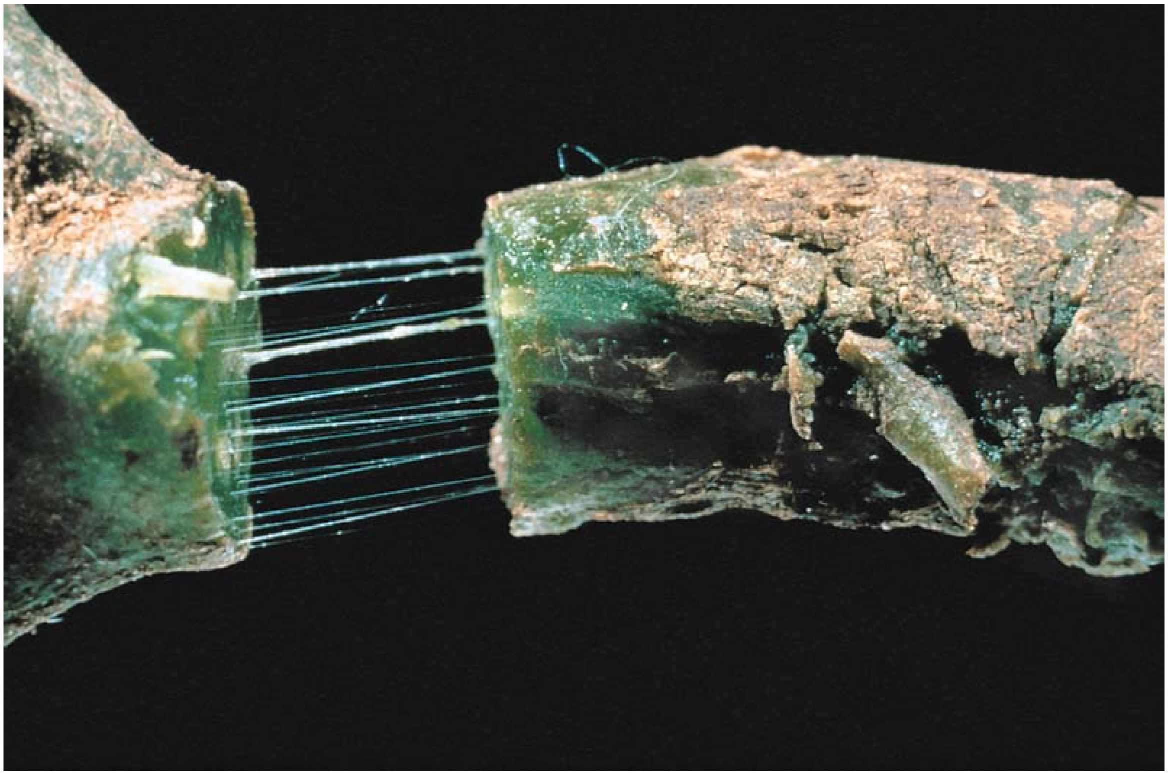


Figure 3.7.
Ooze, a diagnostic sign of a bacterial infection. In bacterial wilt of cucurbits, the sticky strands of bacterial ooze become evident as two portions of a cut stem are pulled apart.

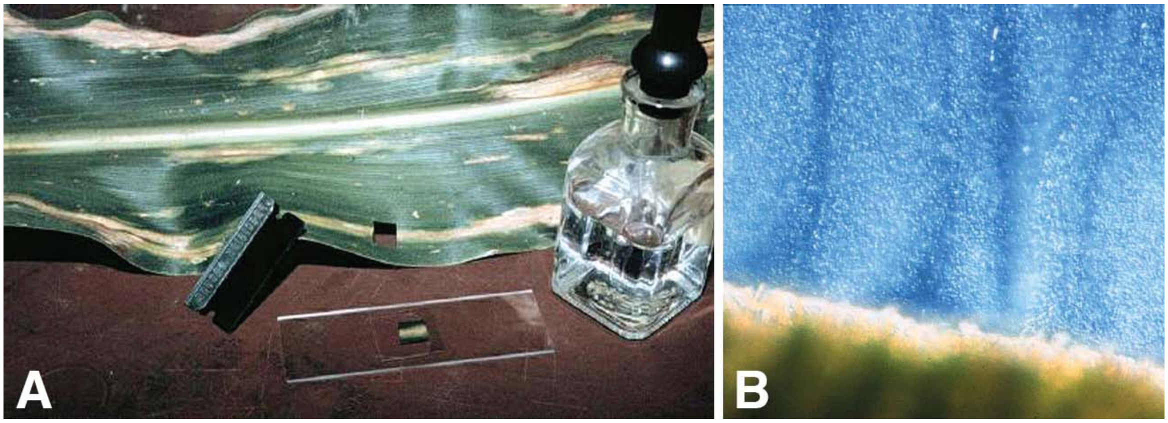


Figure 3.8.
A, To diagnose Stewart’s wilt, a section of symptomatic corn leaf tissue is placed in a drop of water on a microscope slide. **B**, Bacterial streaming, viewed with a compound microscope.

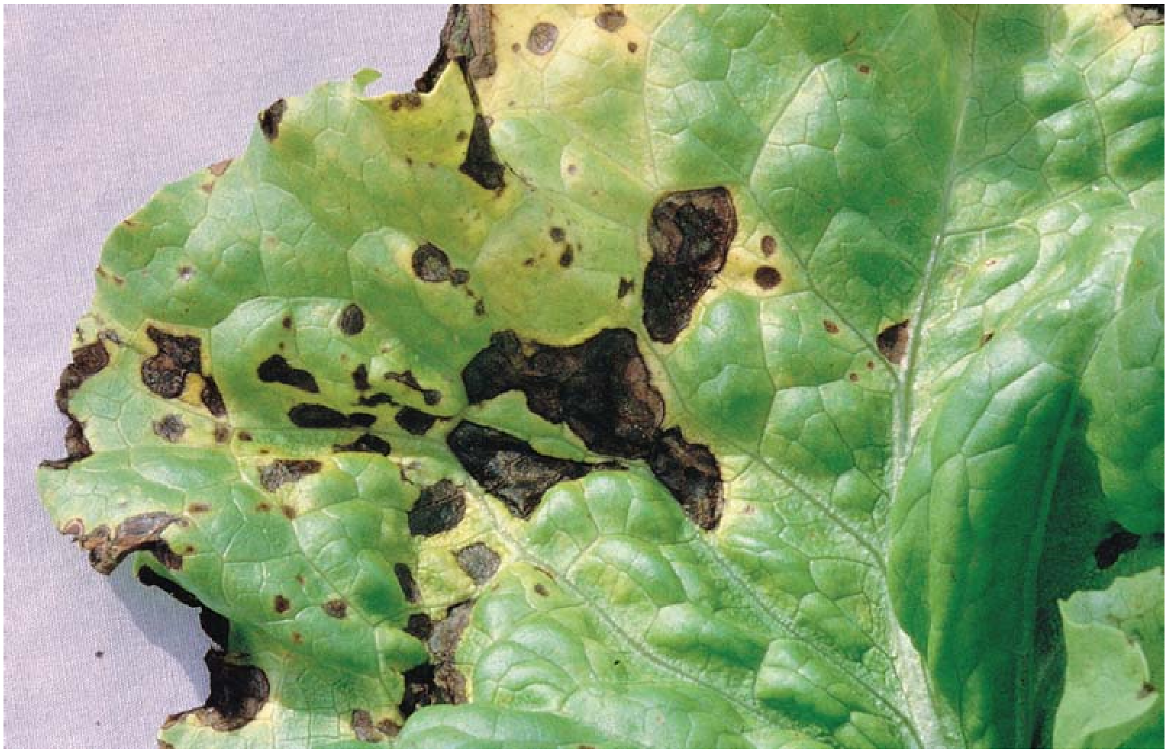


Figure 3.9.
Angular leaf spots typical of those caused by *Pseudomonas* spp. and *Xanthomonas* spp.

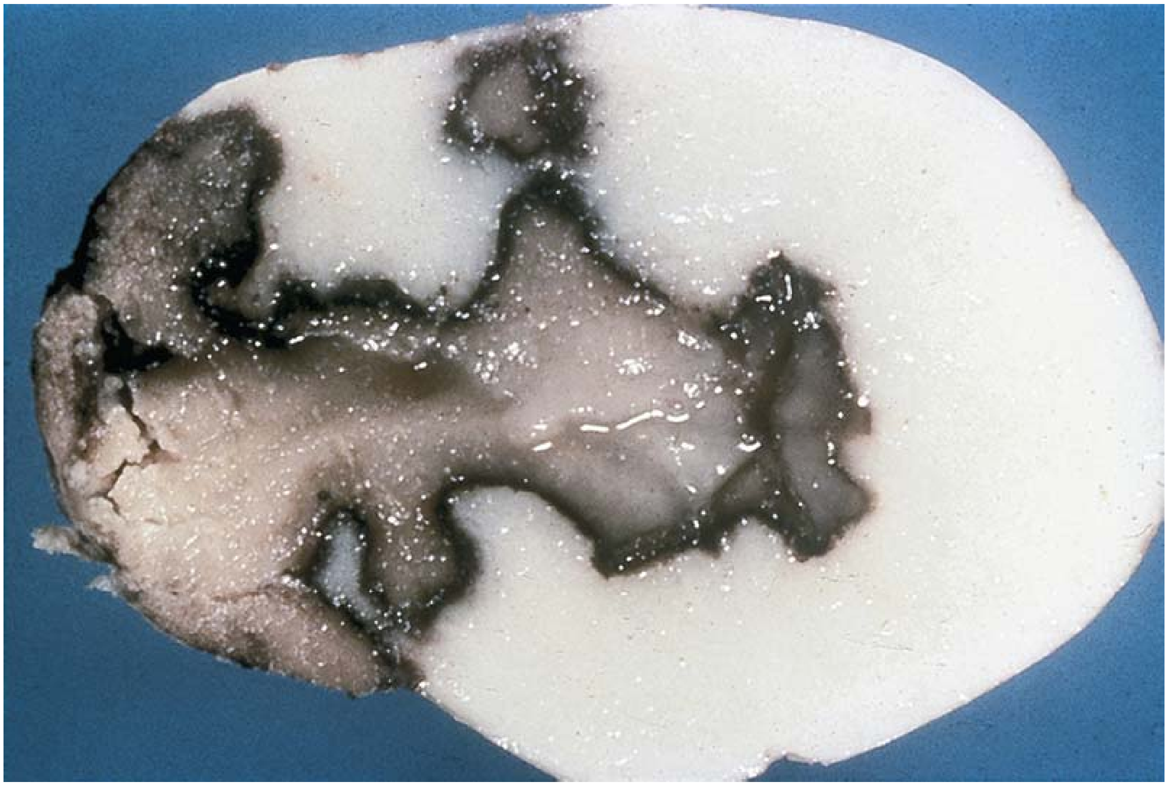


Figure 3.10.
Soft rot in a potato tuber.

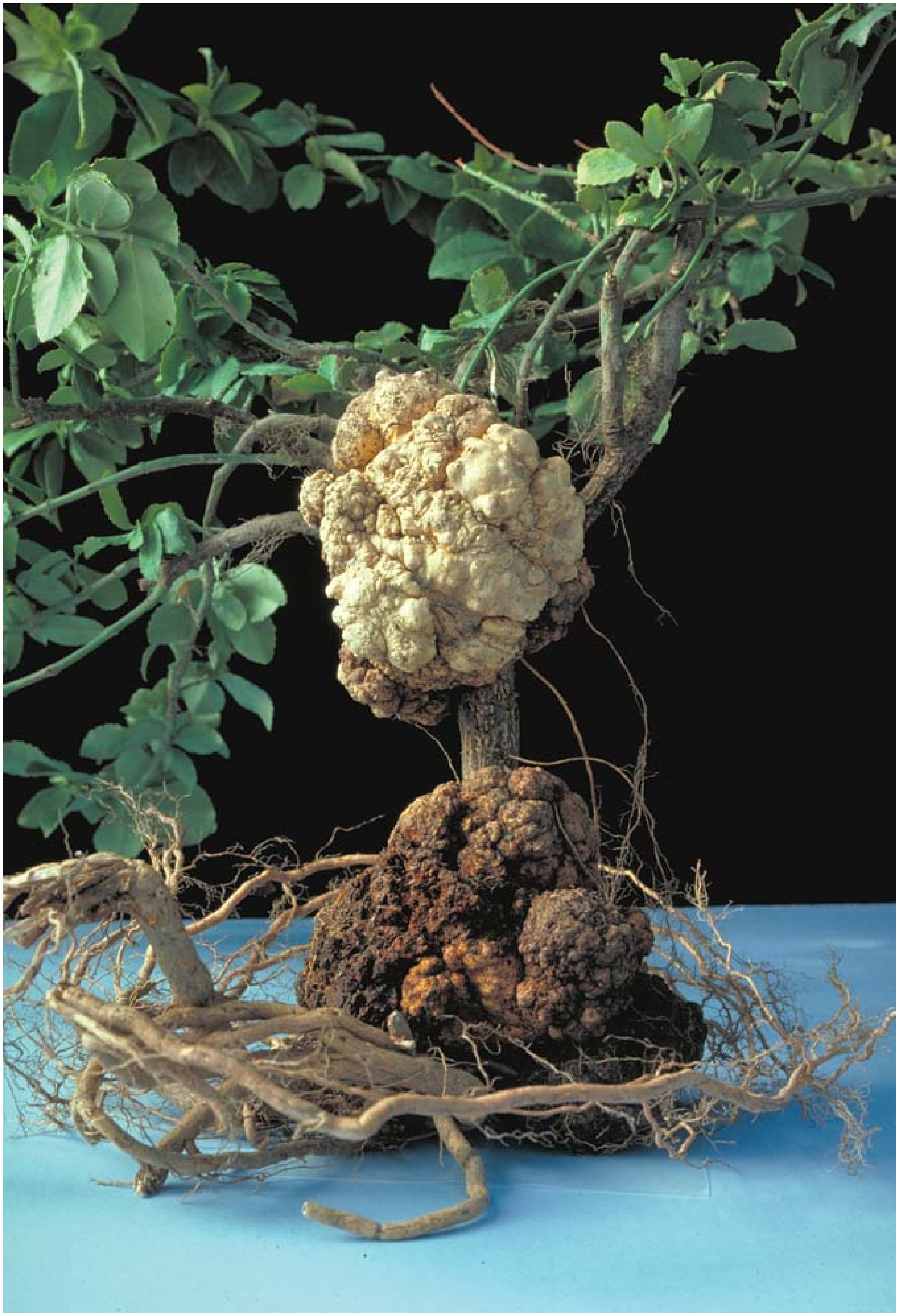


Figure 3.11.
Crown gall on euonymous caused by *Agrobacterium tumefaciens*.

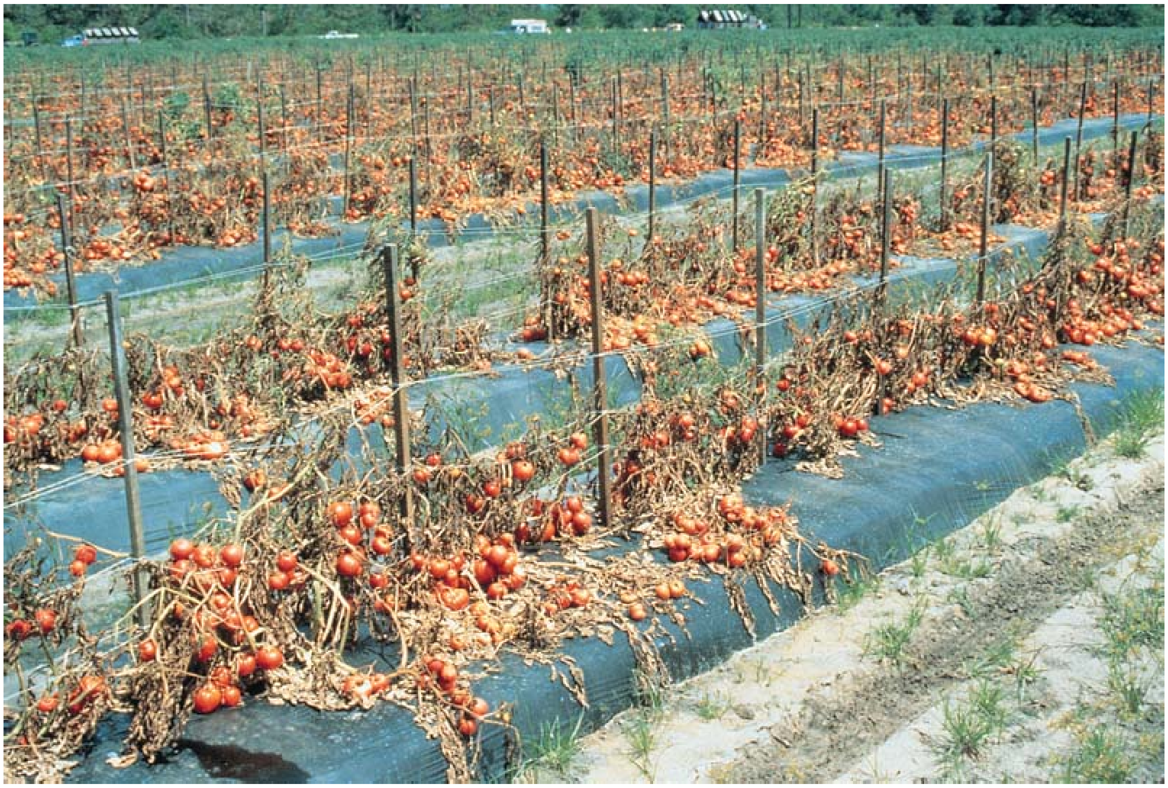


Figure 3.12.
Bacterial wilt of tomatoes caused by *Ralstonia solanacearum*.

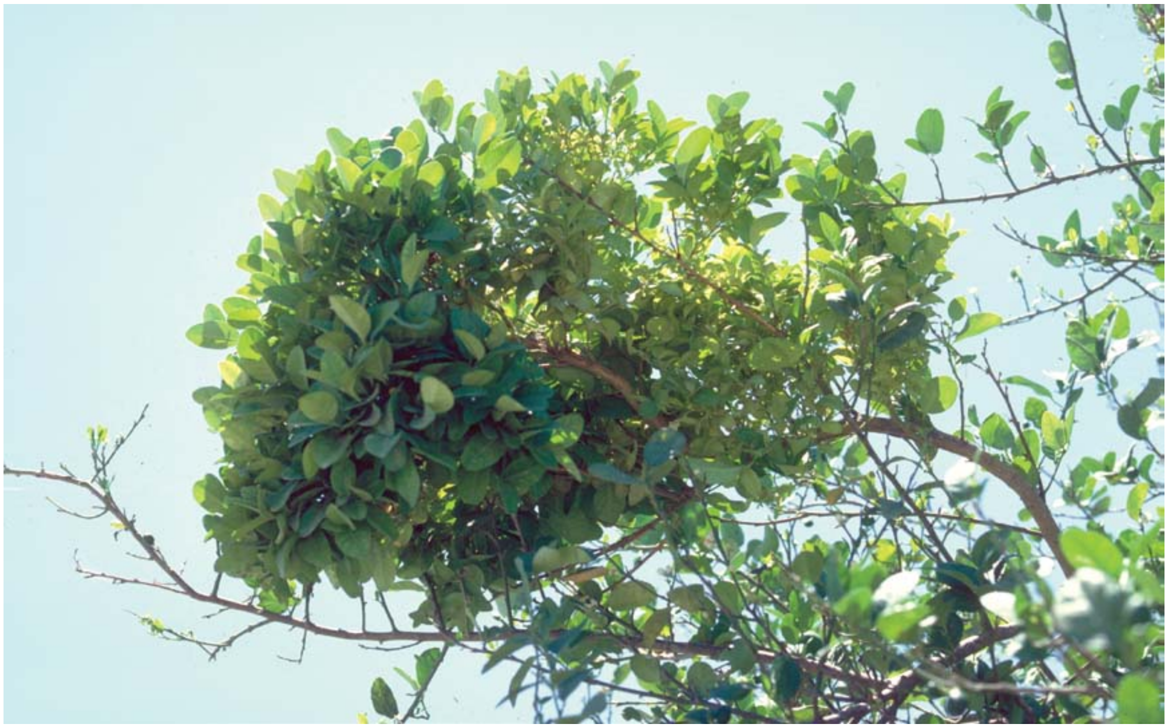


Figure 3.13.
Witches’ brooming in lime infected by a phytoplasma.

Figure 3.14.

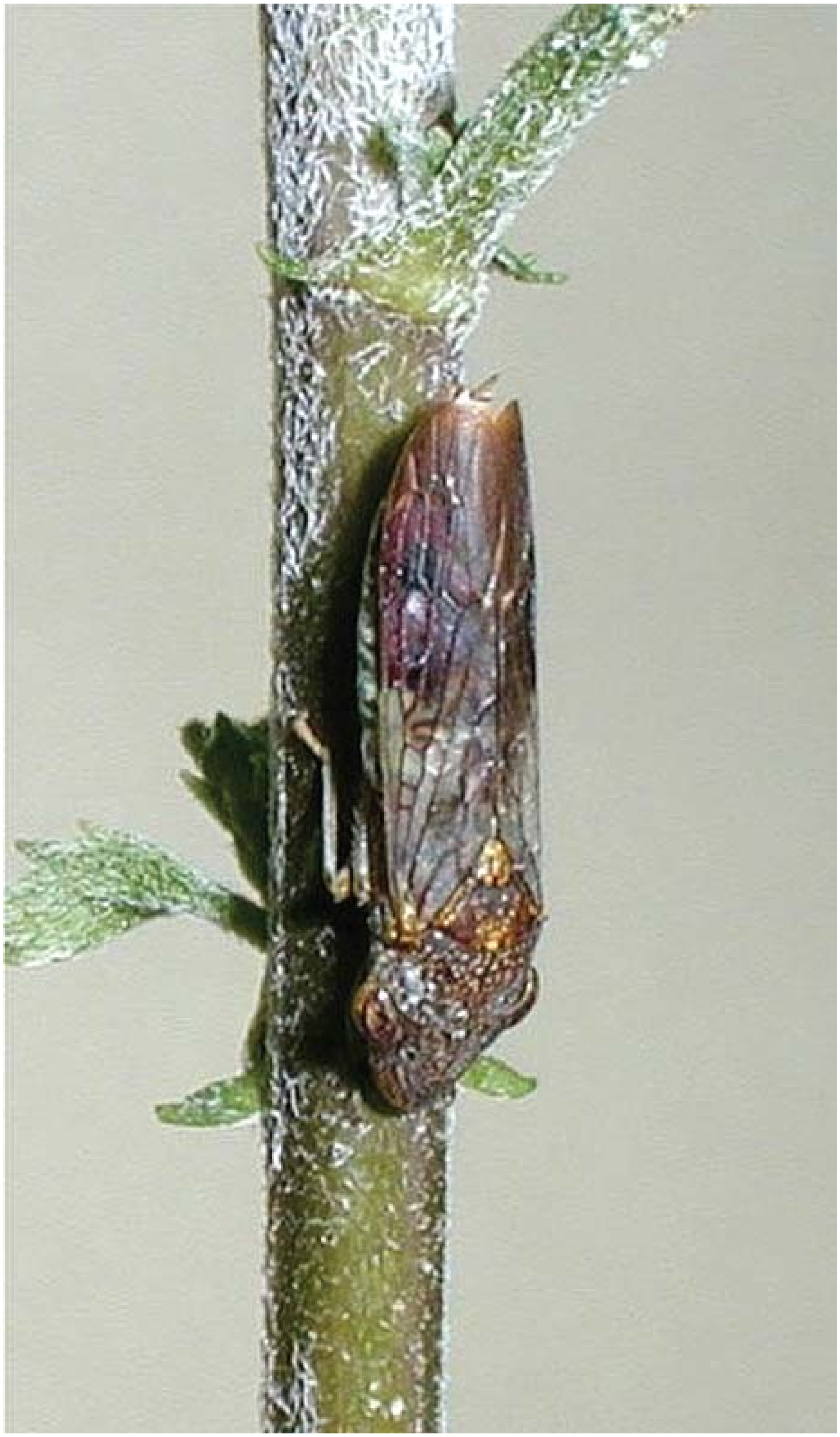


Figure 3.14.

The glassy-winged sharpshooter, a leafhopper vector of *Xylella fastidiosa*. These insects subsist on xylem fluid and often feed upside down.

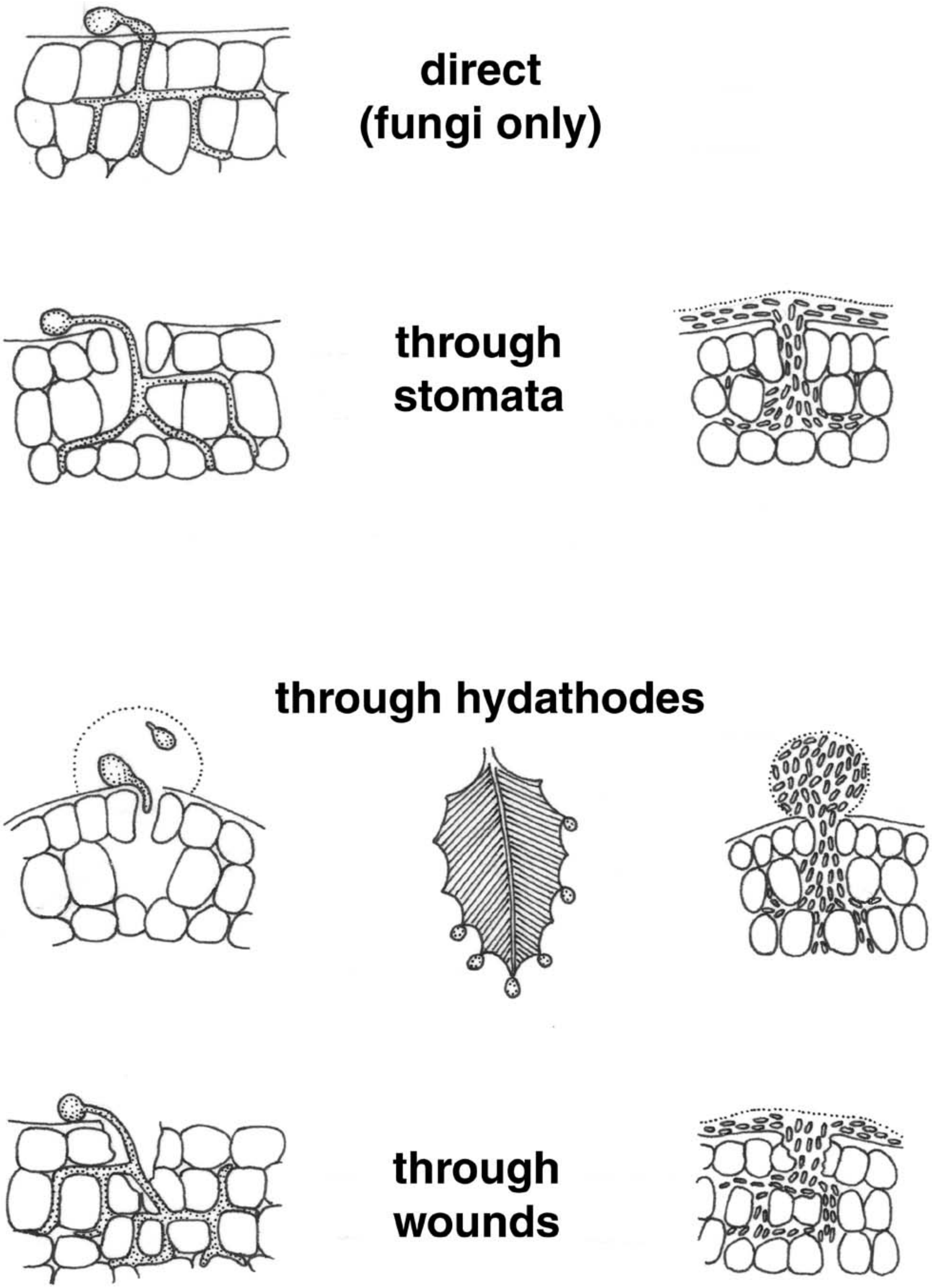

direct
(fungi only)
through
stomata
through hydathodes
through
wounds

Figure 3.15.

Various modes of penetration and invasion of a leaf by fungi (left) and bacteria (right). Water is required for bacteria to enter cells.

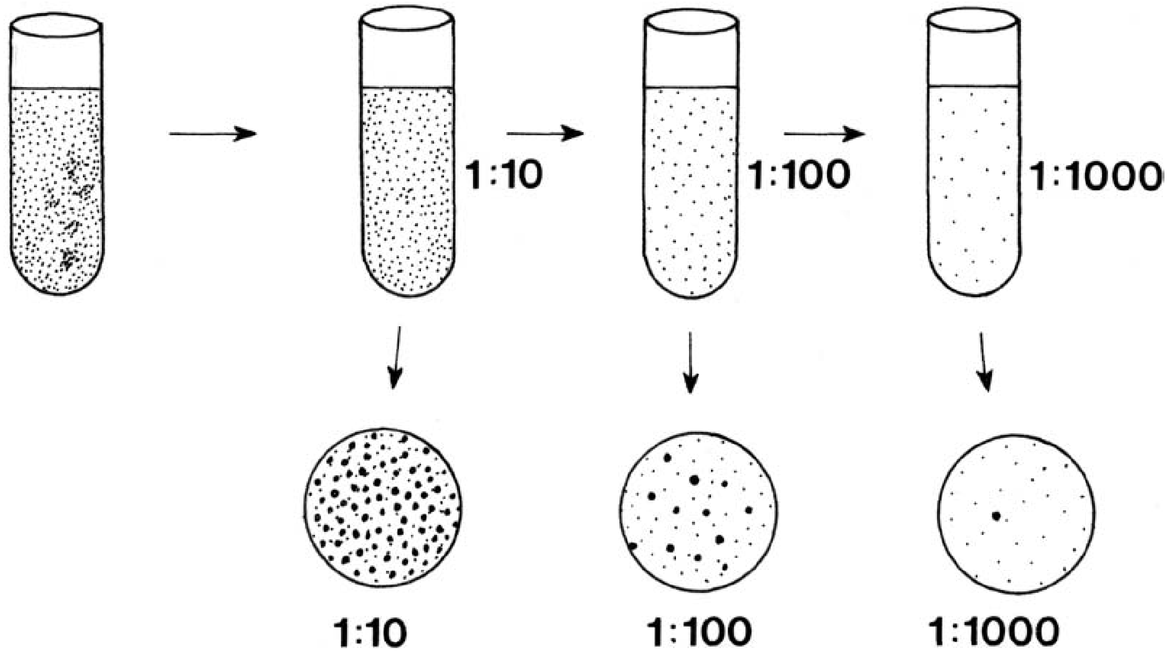

1:10
1:100
1:1000
1:10
1:100
1:1000

Figure 3.16.
Dilution plating to obtain pure colonies of a bacterium. The tissue is torn apart to release the bacteria. The sample is then diluted to reduce background contaminants. A sample of each dilution is spread on a plate of nutrient medium.

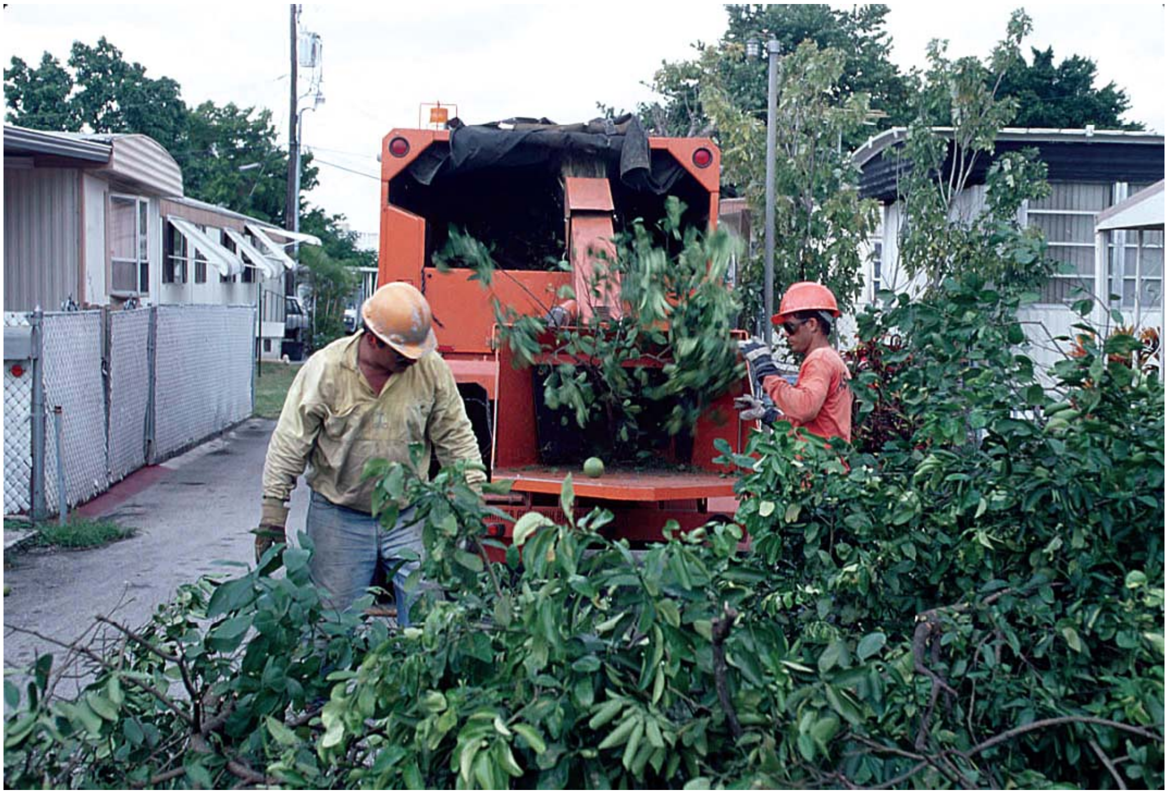


Figure 3.17.

Commercial chipping machine used to chip citrus trees infected by or exposed to citrus canker in urban Miami, Florida.

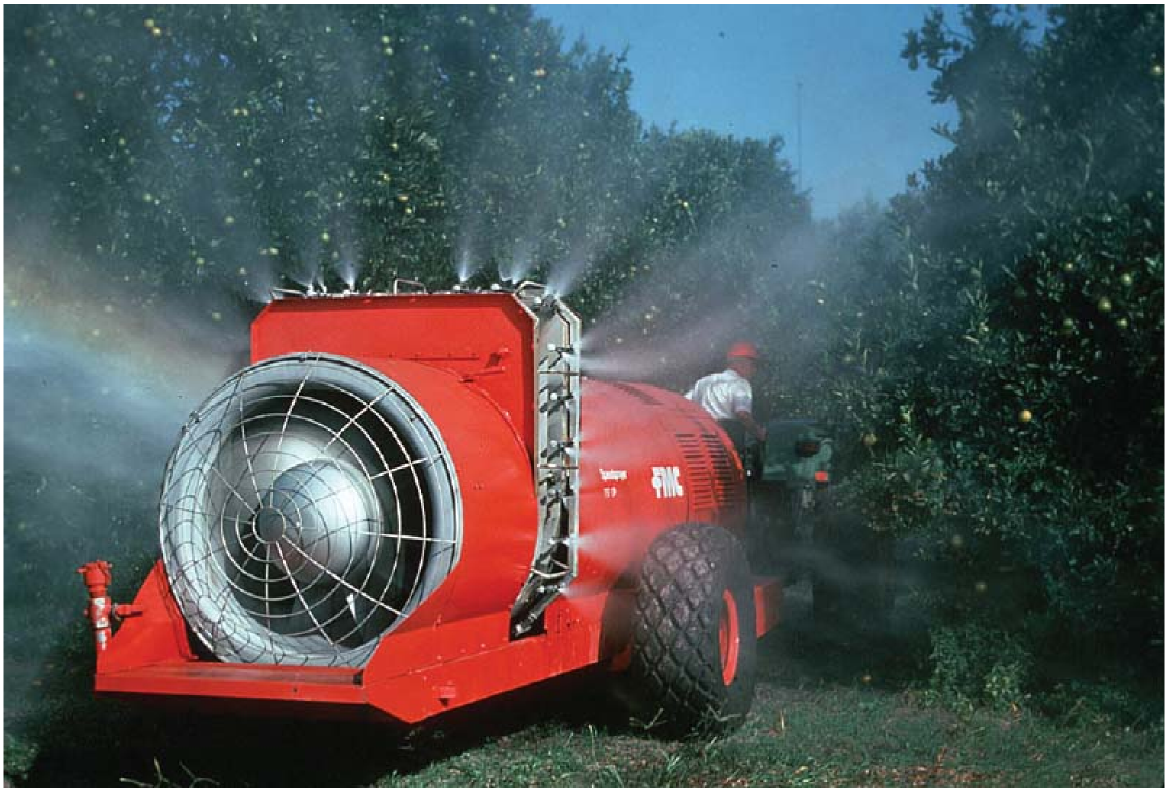


Figure 3.18.
Typical air-blast sprayer for application of copper-based sprays or antibiotics.

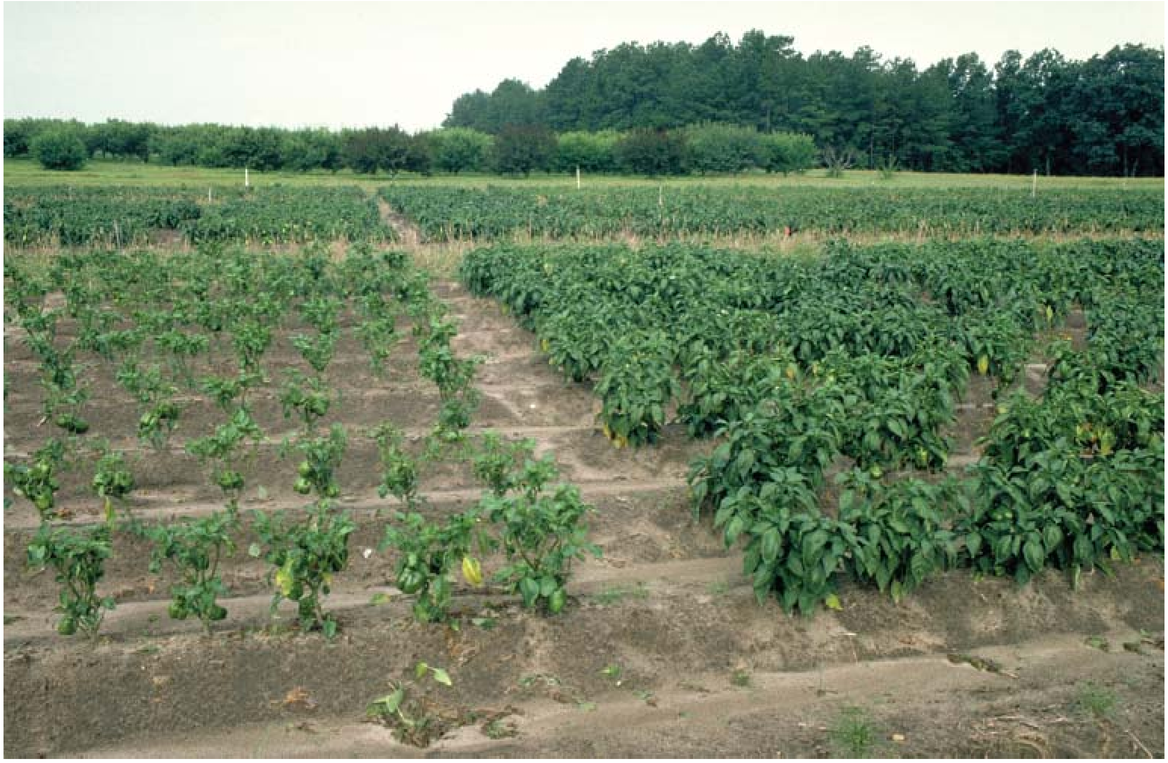


Figure 3.19.
Pepper plants susceptible (left) and resistant (right) to specific strains of the leaf spot bacterium, *Xanthomonas axonopodis* pv. *vesicatoria*.

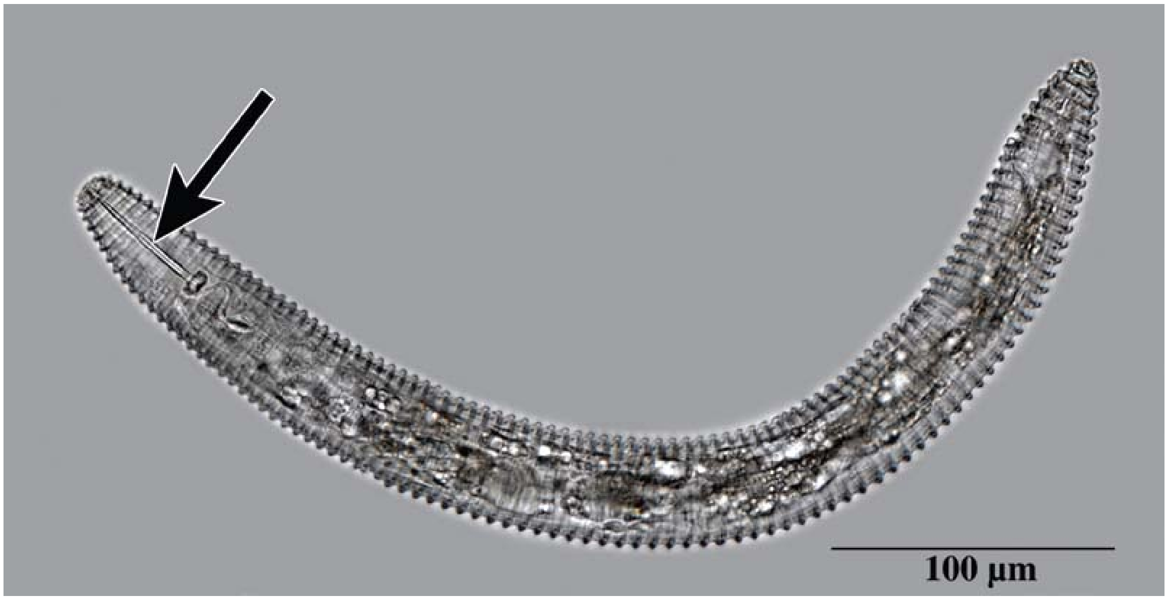


Figure 4.1.

A ring nematode (*Mesocriconema xenoplax*). Note the stylet, the feeding structure (arrow).

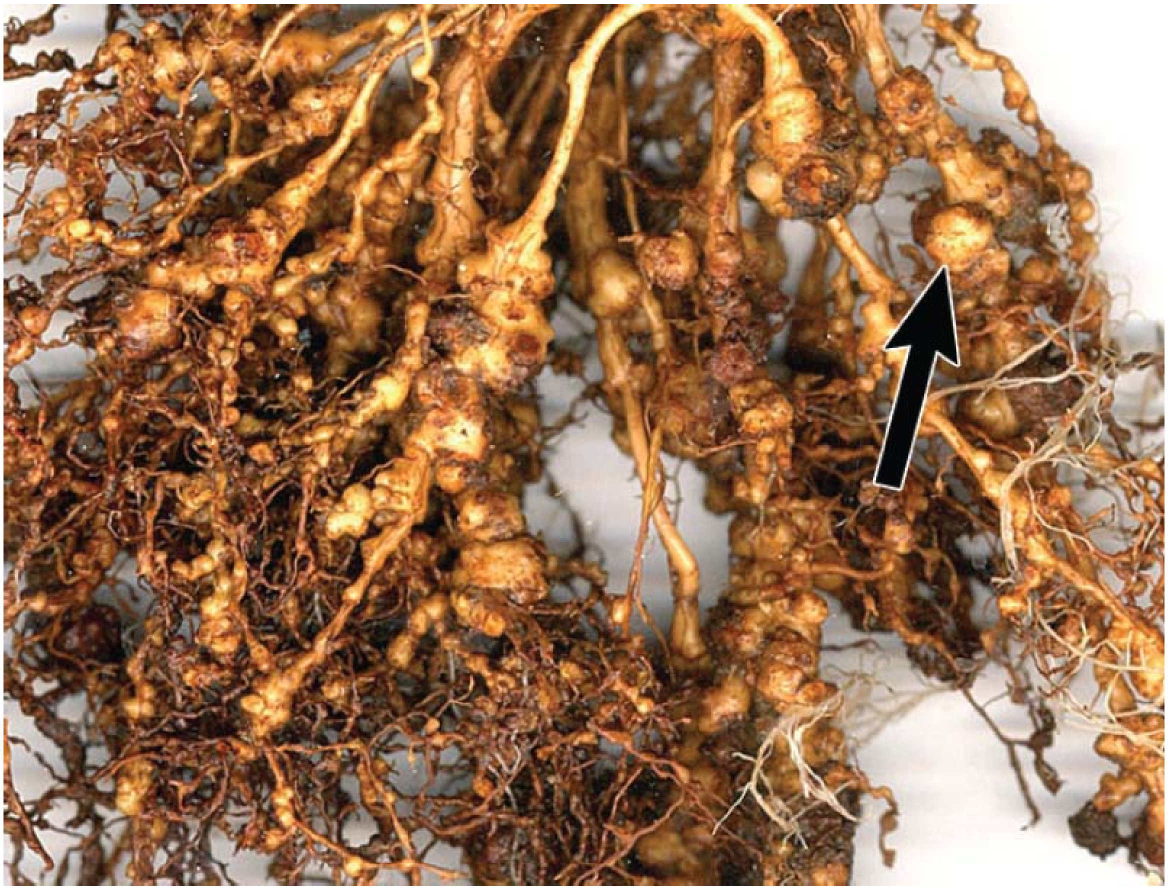


Figure 4.2.

Closeup of root knots (galls) (arrow) on lettuce caused by root-knot nematodes (*Meloidogyne* spp.).

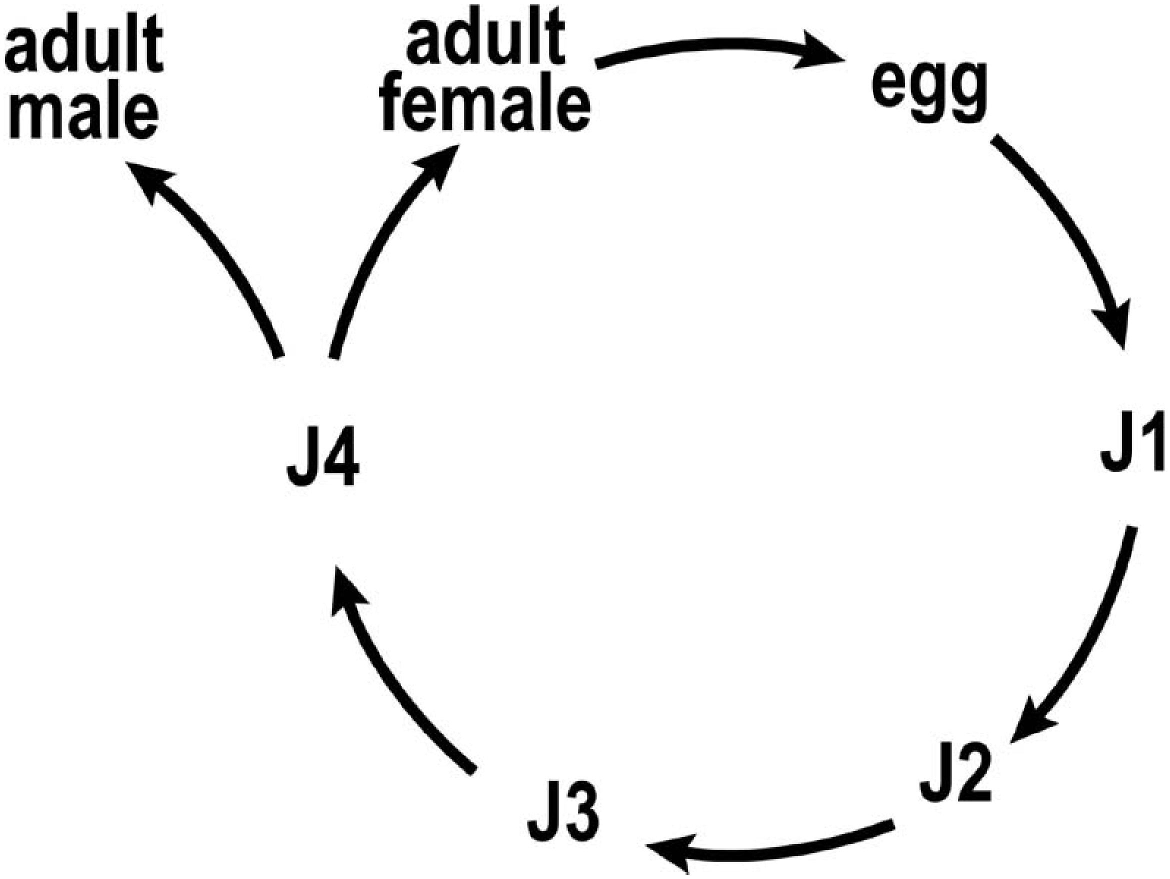
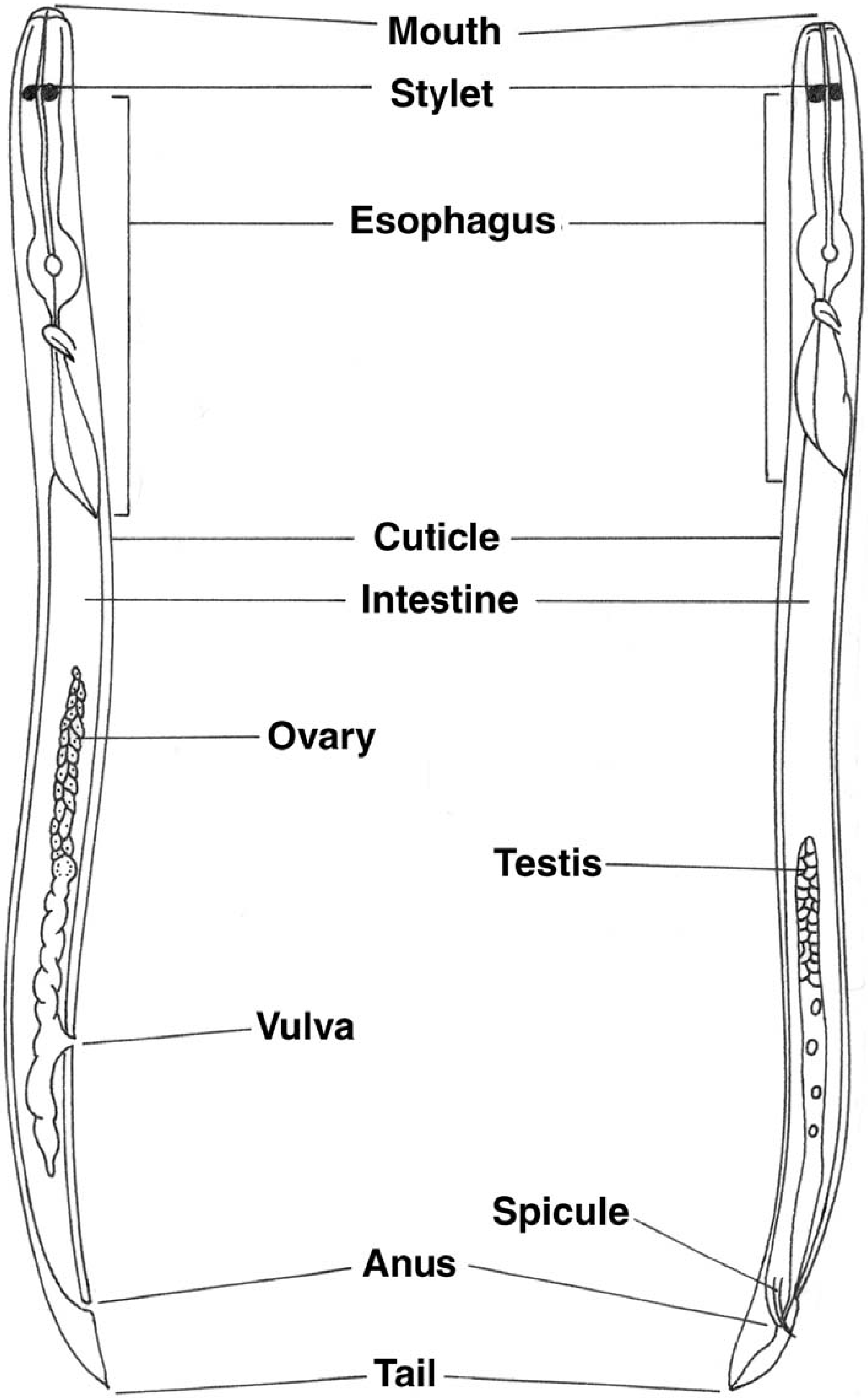

adult male
adult female
egg
J1
J2
J3
J4

Figure 4.3.
Life cycle of a nematode including four juvenile (J1-J4) stages.


Mouth
Stylet
Esophagus
Cuticle
Intestine
Ovary
Testis
Vulva
Spicule
Anus
Tail

Female Male

Figure 4.4.
Generalized nematode anatomy.

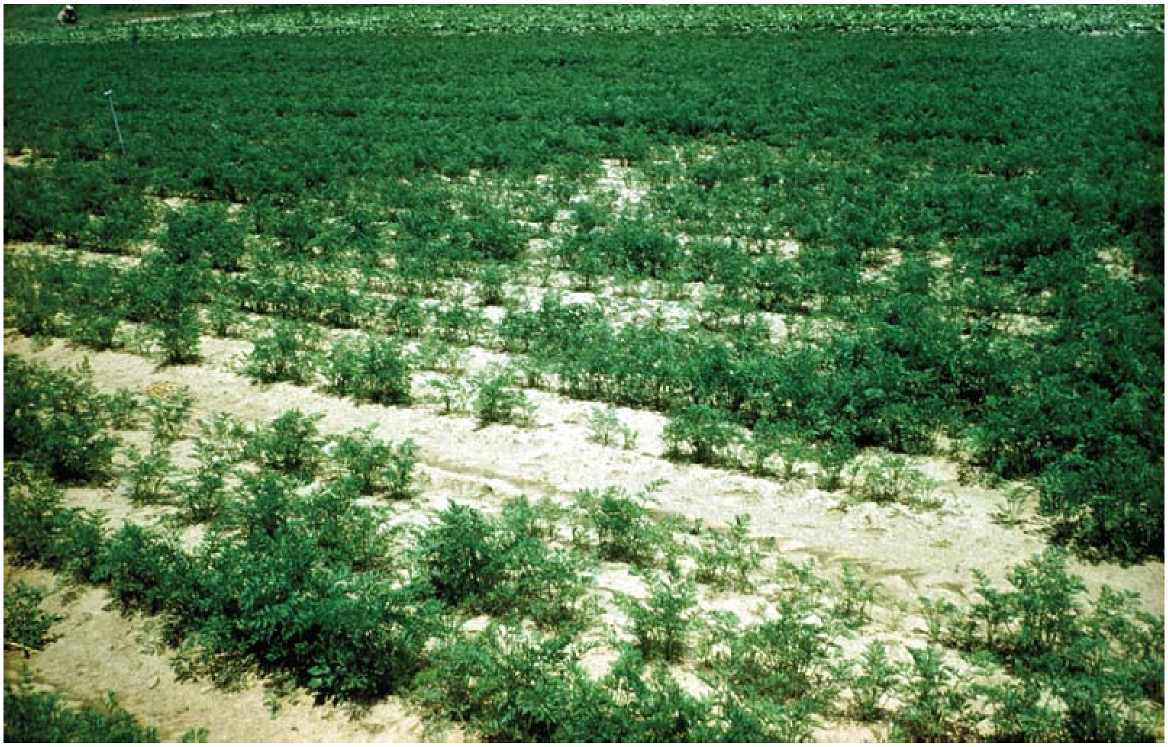


Figure 4.5.
Typical symptoms of nematode damage in the field include stunted plants in a somewhat circular pattern (or “hot spot”). (These are carrots infected by lesion nematodes.)

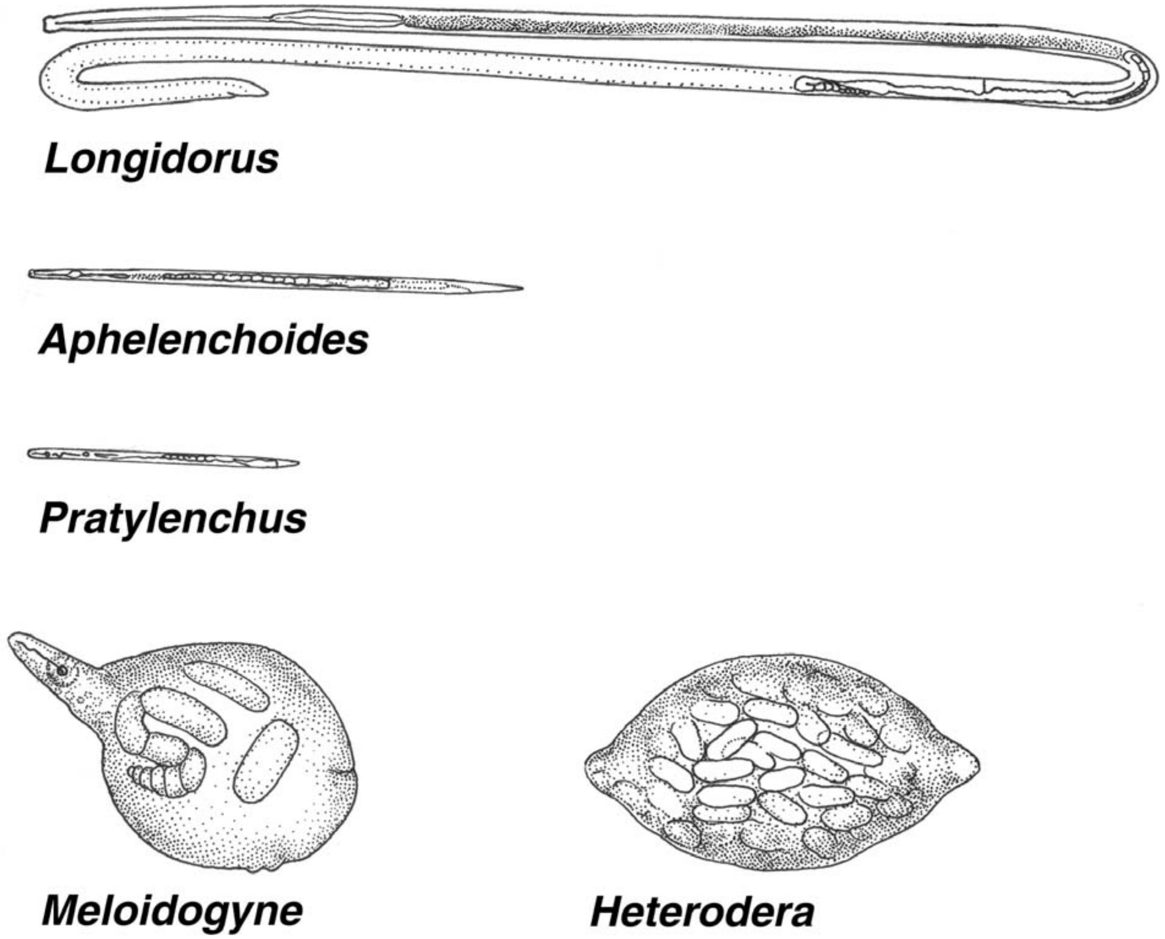

Longidorus
Aphelenchoides
Pratylenchus
Meloidogyne
Heterodera

Figure 4.6.
Morphology and relative size of some important plant-pathogenic nematodes.

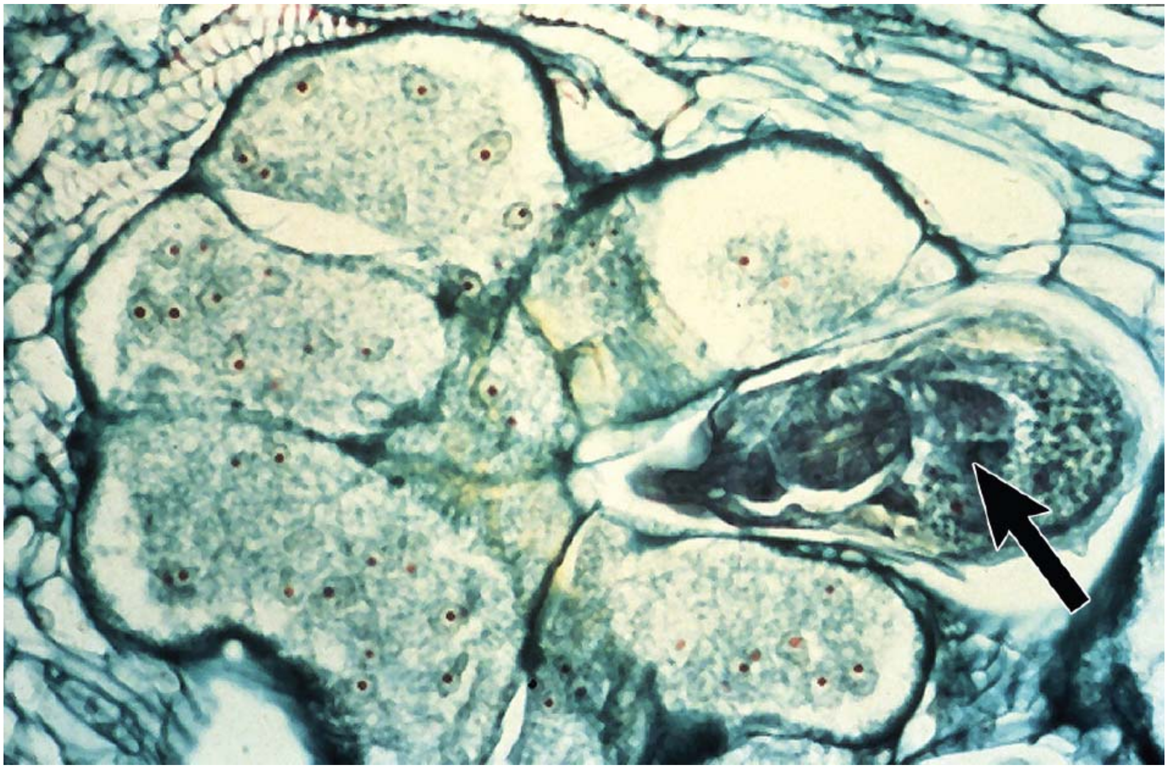


Figure 4.7.
Cross section of the giant cell system initiated by a female root-knot nematode (arrow).

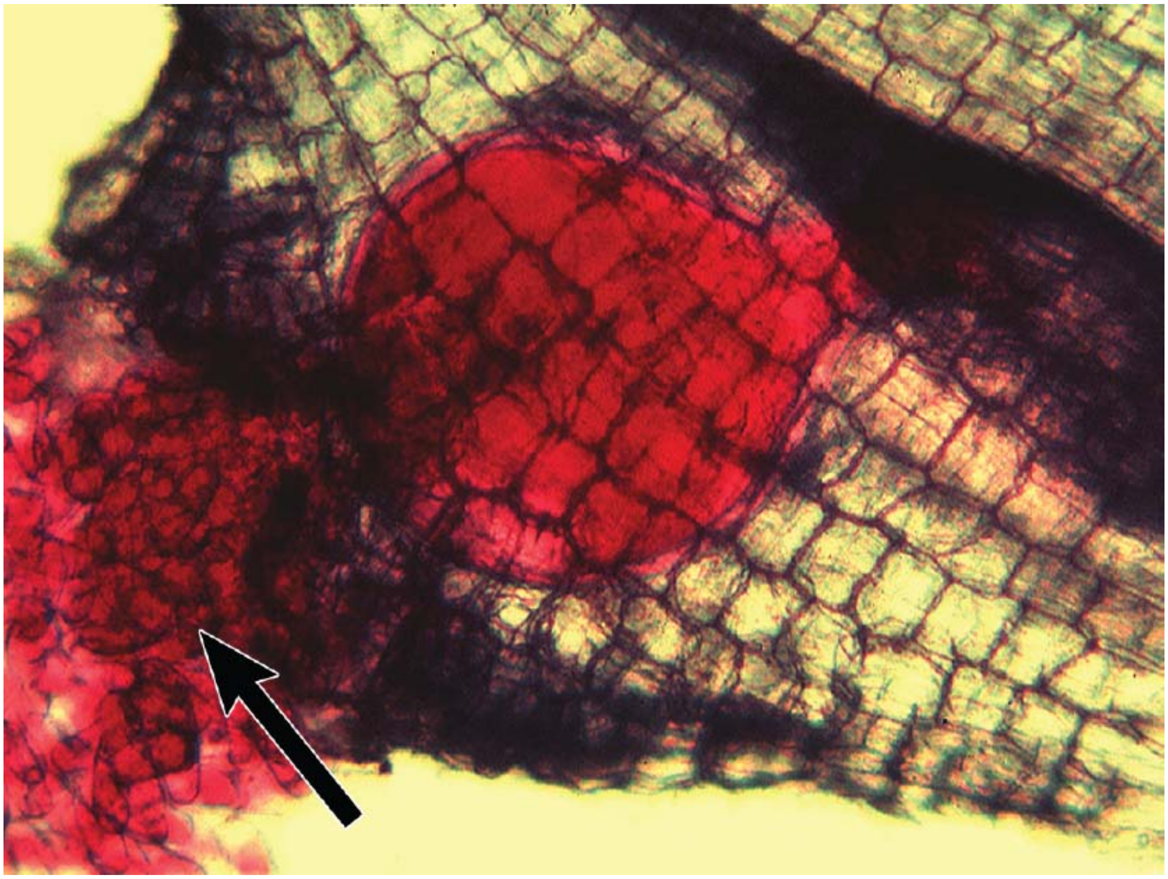


Figure 4.8.
Root-knot nematode female with egg mass (arrow).

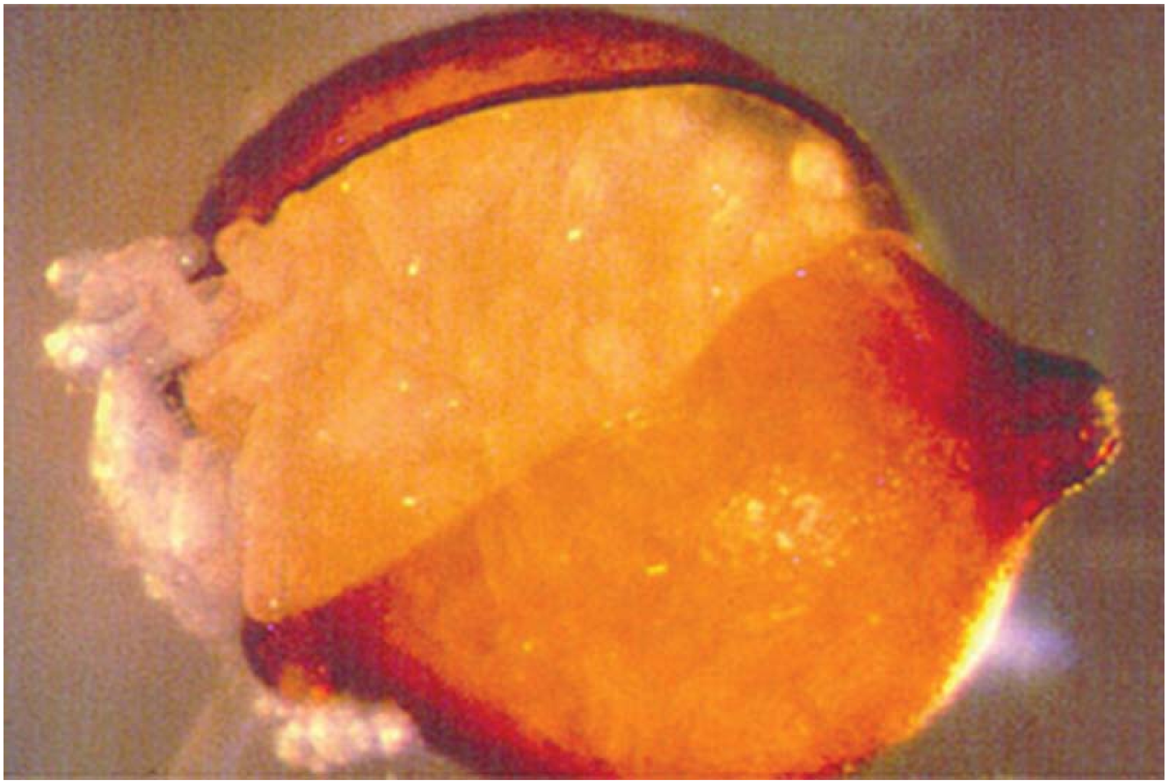


Figure 4.9.
Brown cyst of a soybean cyst nematode filled with eggs.

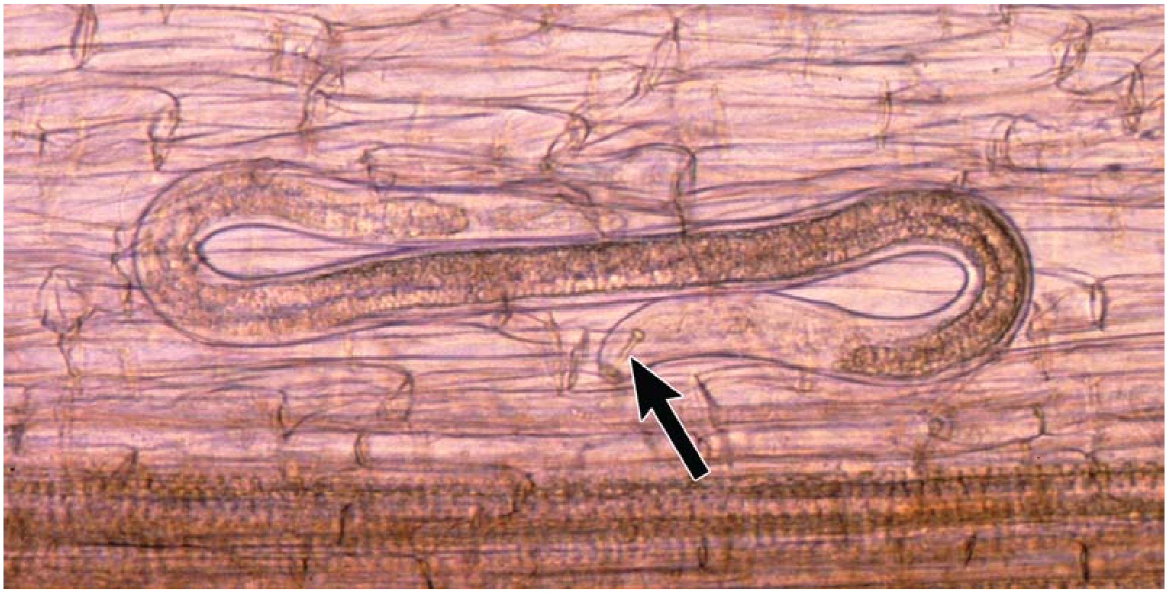


Figure 4.10.
Lesion nematode (*Pratylenchus* sp.) in a plant root. Note the stylet, the feeding structure (arrow).

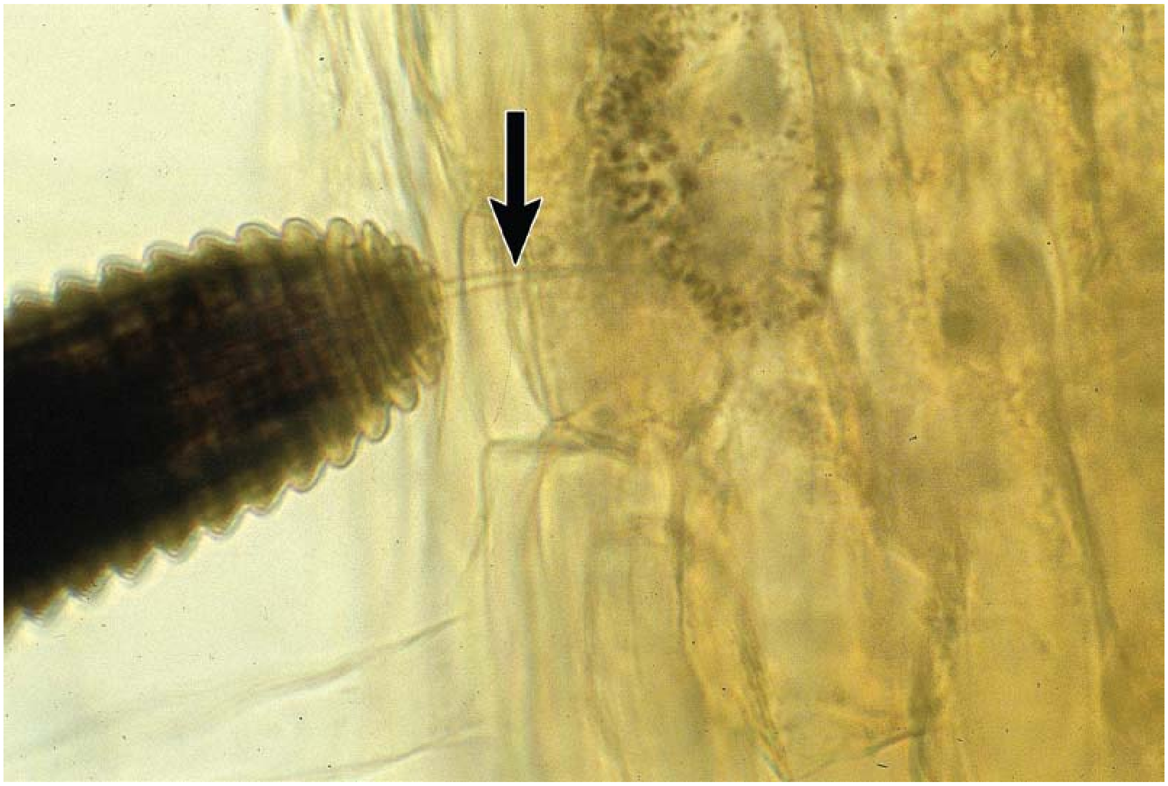


Figure 4.11.
An ectoparasitic ring nematode (*Mesocriconema xenoplax*) feeding on a root. Note stylet (arrow).

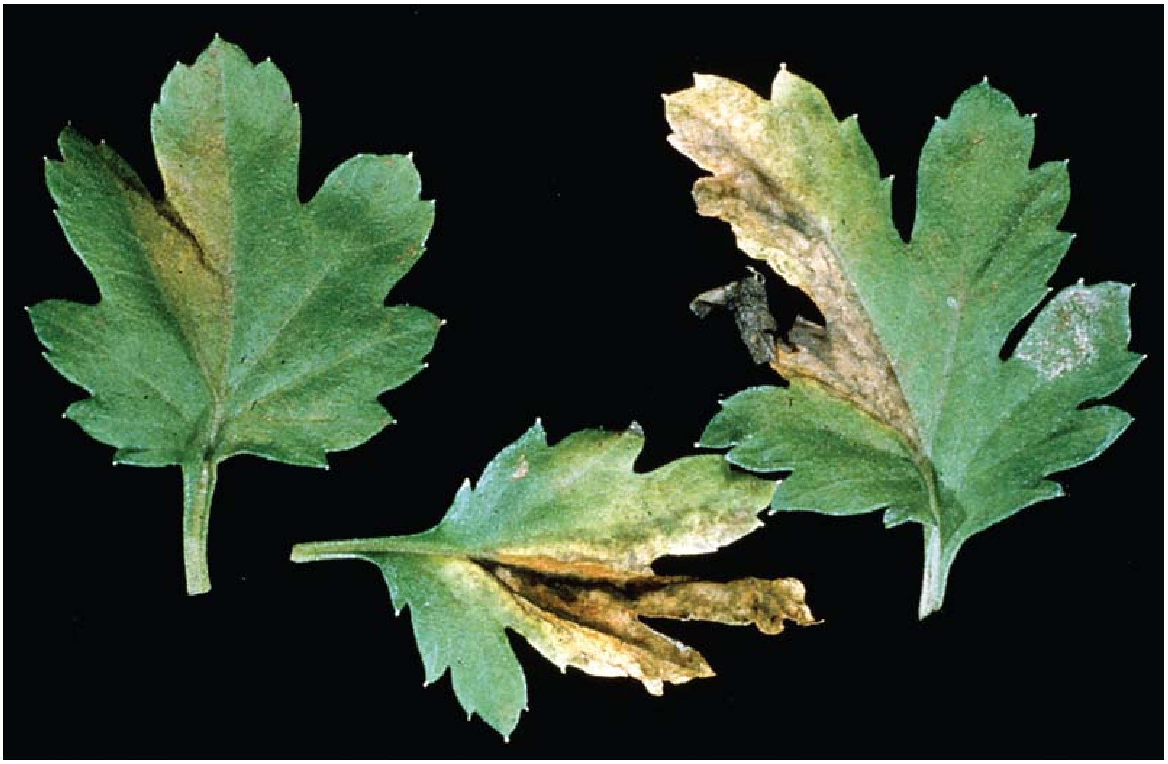


Figure 4.12.
Feeding damage on chrysanthemum from foliar nematodes (*Aphelenchoides* spp.). Note leaf lesions bound by major leaf veins.

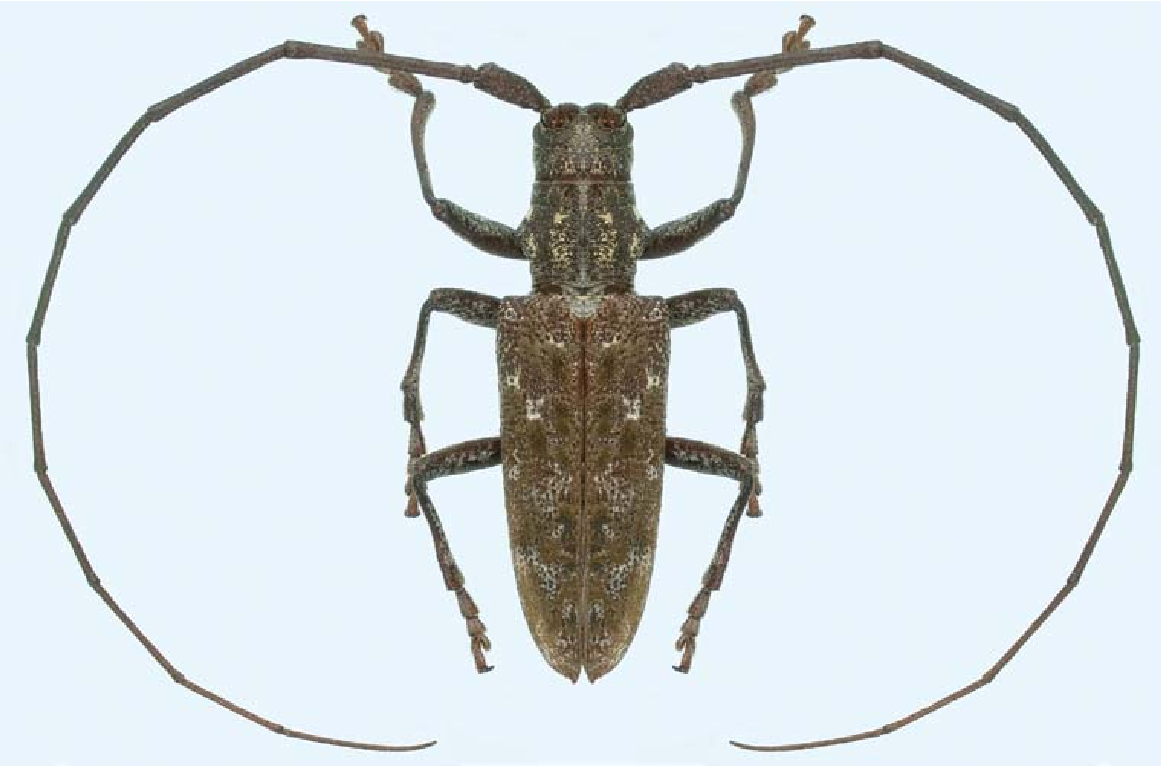


Figure 4.13.
Pine sawyer beetle (*Monochamus carolinensis*), vector of the pine wood nematode (*Bursaphelenchus xylophilus*), which causes pine wilt disease.

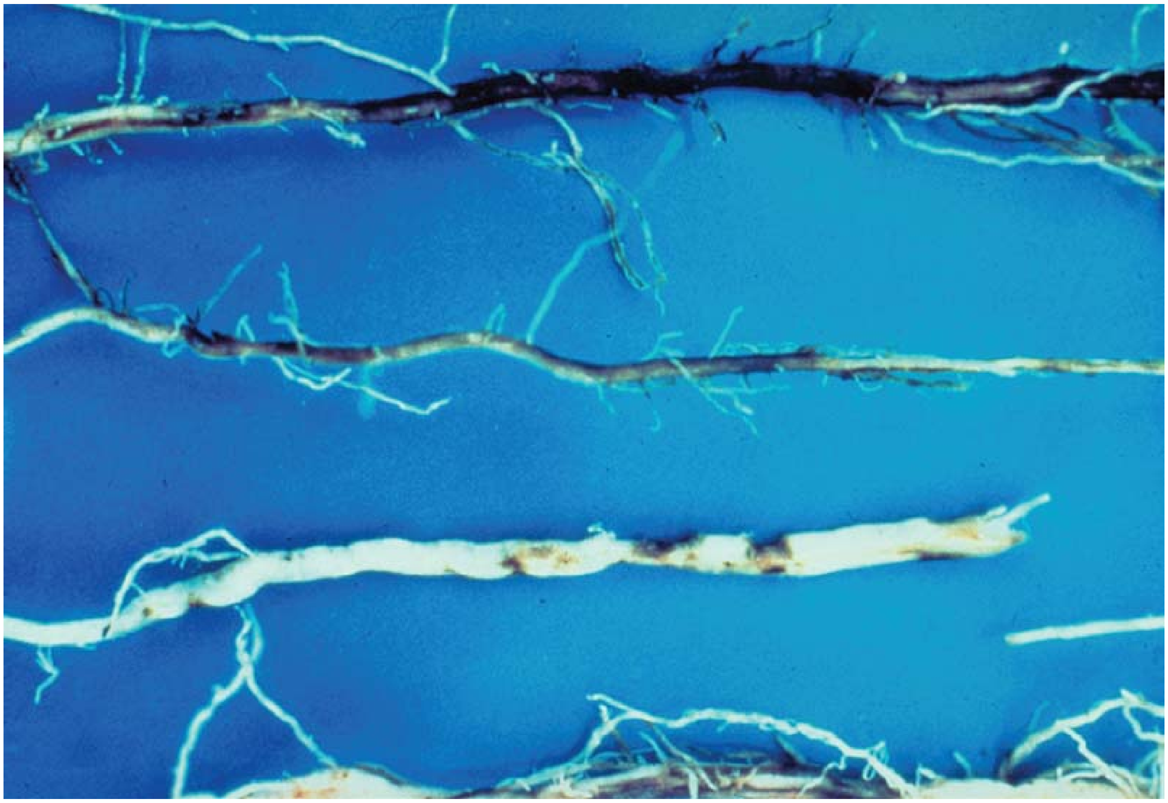


Figure 4.14.
Necrotic lesions on corn roots infected by lesion nematodes (*Pratylenchus* spp.).

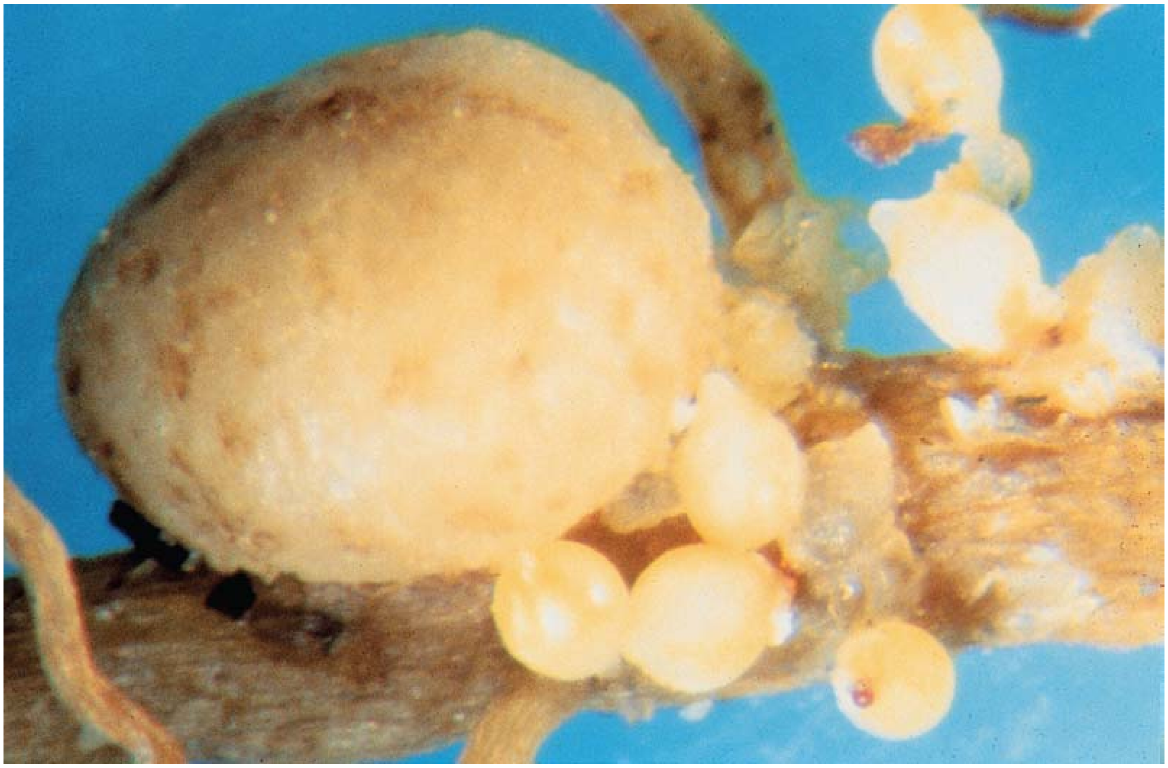


Figure 4.15.
Soybean cyst nematode females (which appear as small, whitish-yellow bodies) erupting through the soybean root surface. The large nodule contains nitrogen-fixing bacteria.

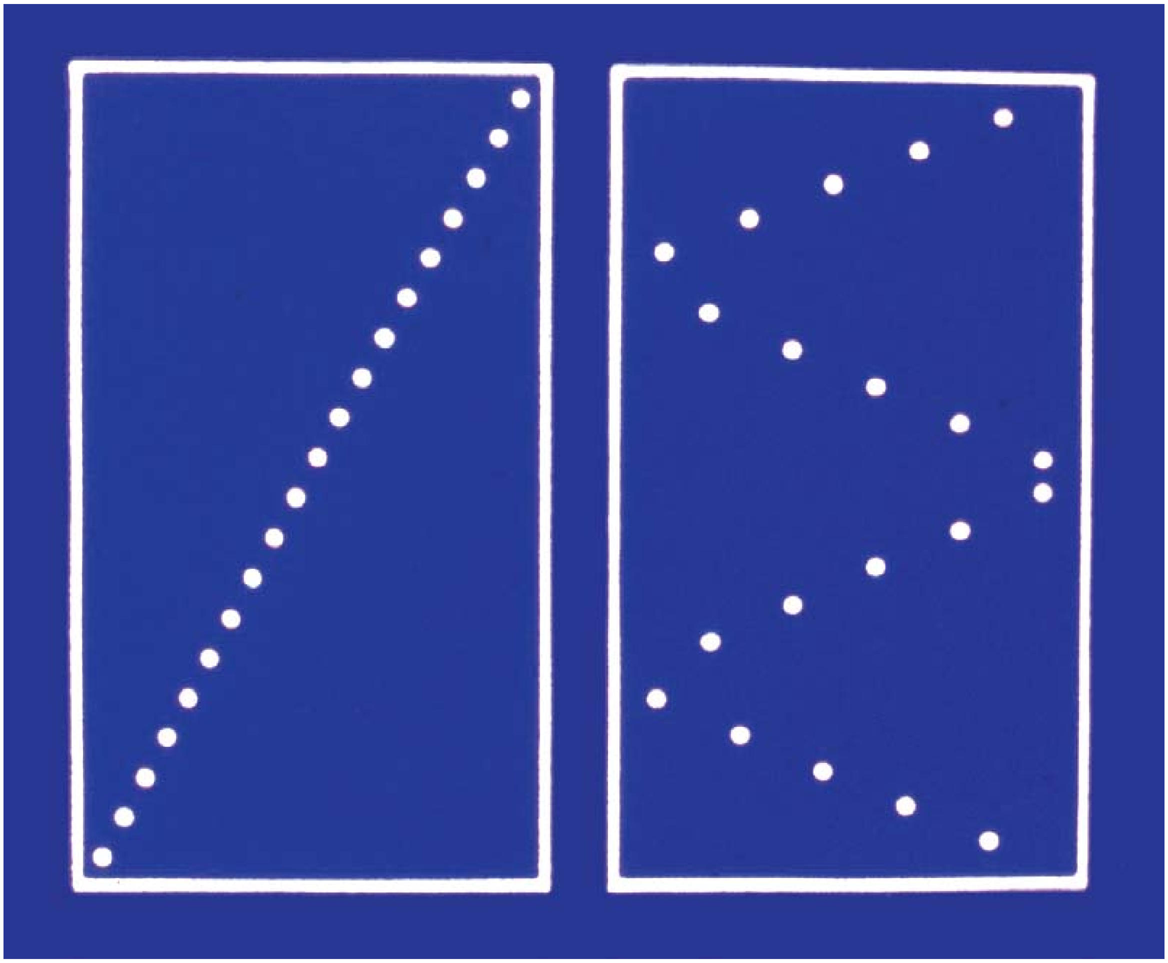


Figure 4.16.
Sampling patterns for assessment of nematode populations.

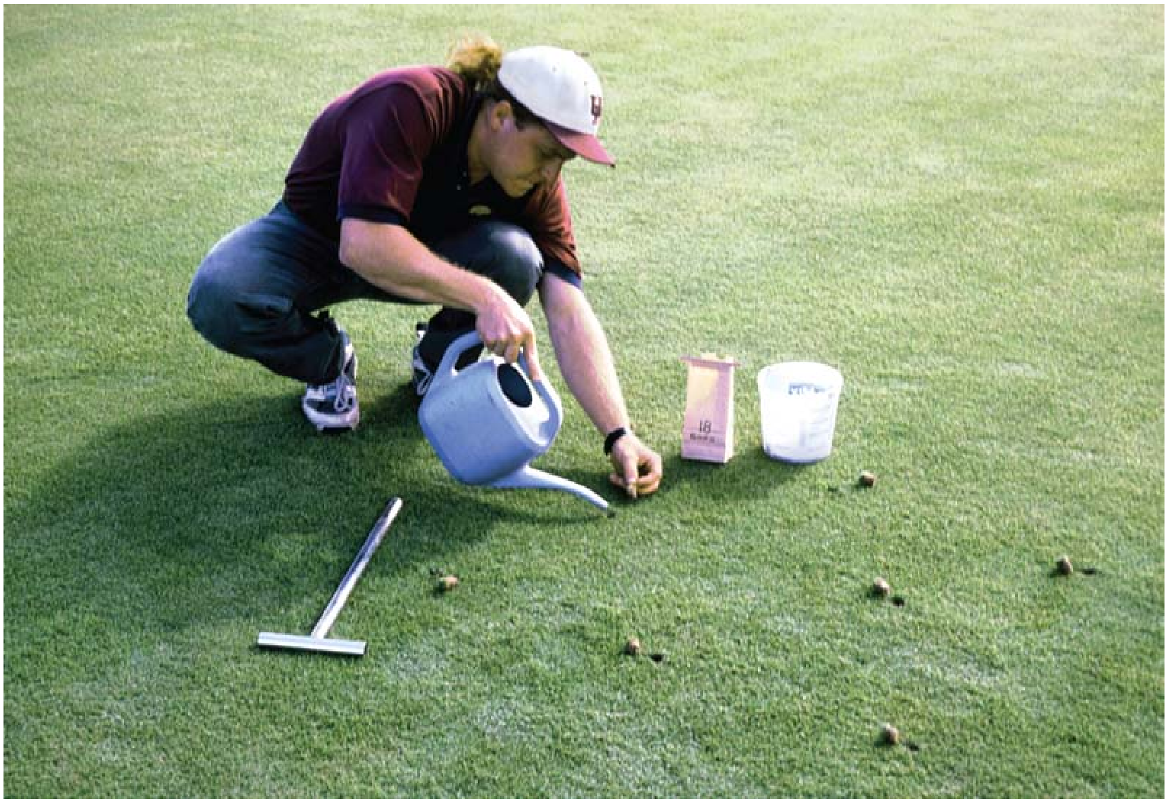
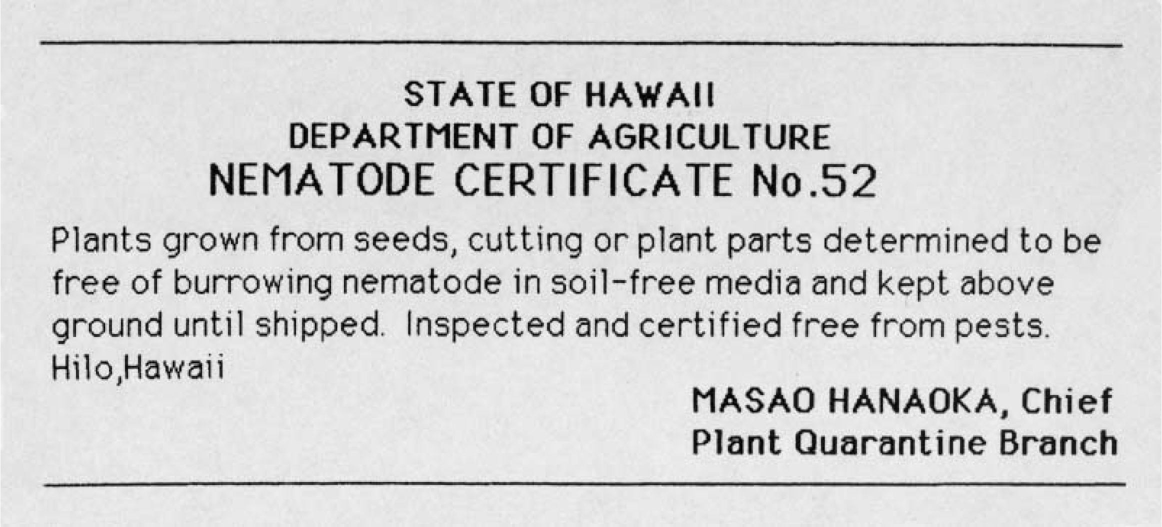


Figure 4.17.
Sampling for nematodes on a golf putting green and filling the holes with sand.



STATE OF HAWAII
DEPARTMENT OF AGRICULTURE
NEMATODE CERTIFICATE No.52

Plants grown from seeds, cutting or plant parts determined to be free of burrowing nematode in soil-free media and kept above ground until shipped. Inspected and certified free from pests.

Hilo,Hawaii

MASAO HANAOKA, Chief
Plant Quarantine Branch

Figure 4.18.
A phytosanitary permit used as part of a program to exclude nematode pathogens. These certificates, issued by the country or state of origin, state that the plants are pathogen-free.

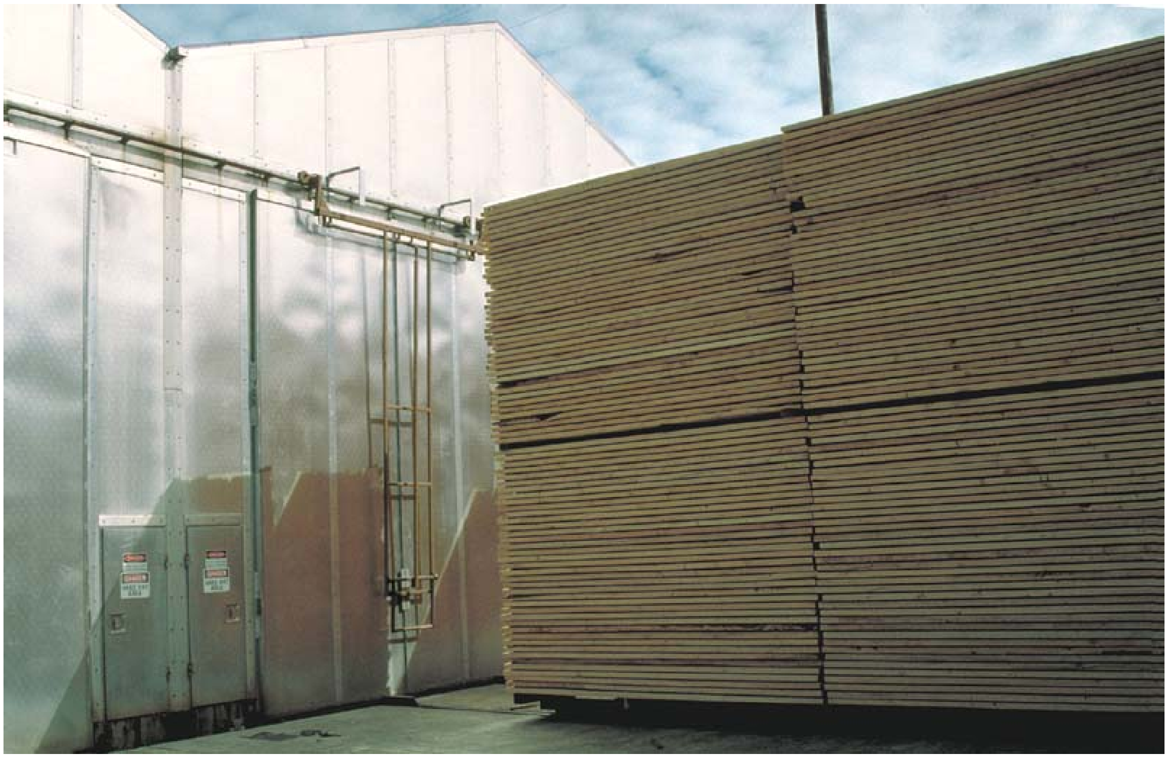


Figure 4.19.
Lumber ready for kiln drying to eradicate pine wood nematodes before export to countries without this pathogen.

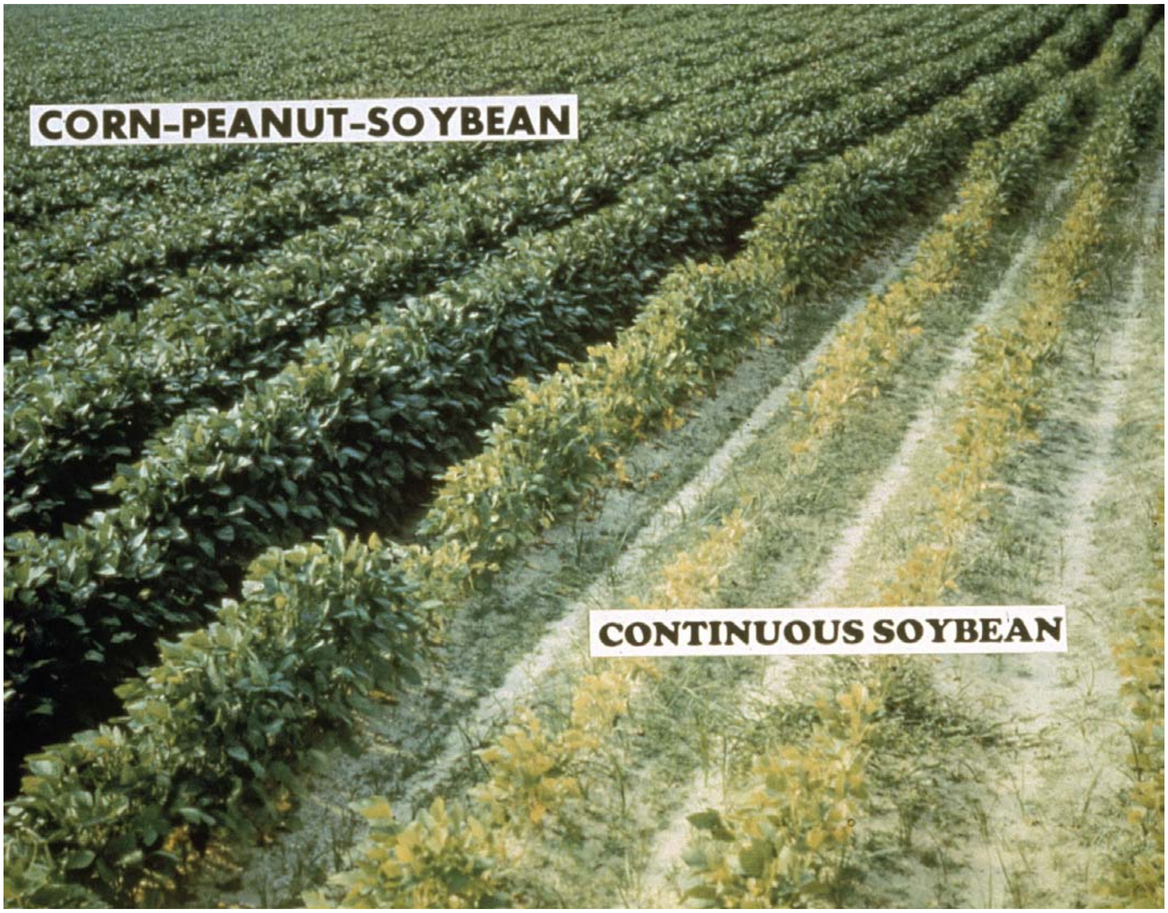

CORN-PEANUT-SOYBEAN
CONTINUOUS SOYBEAN

Figure 4.20.
Field showing reduction in soybean cyst nematode damage to soybean in areas (left) where corn and peanut (nonhosts) were rotated with soybean. The area on the right was planted to soybean continuously.

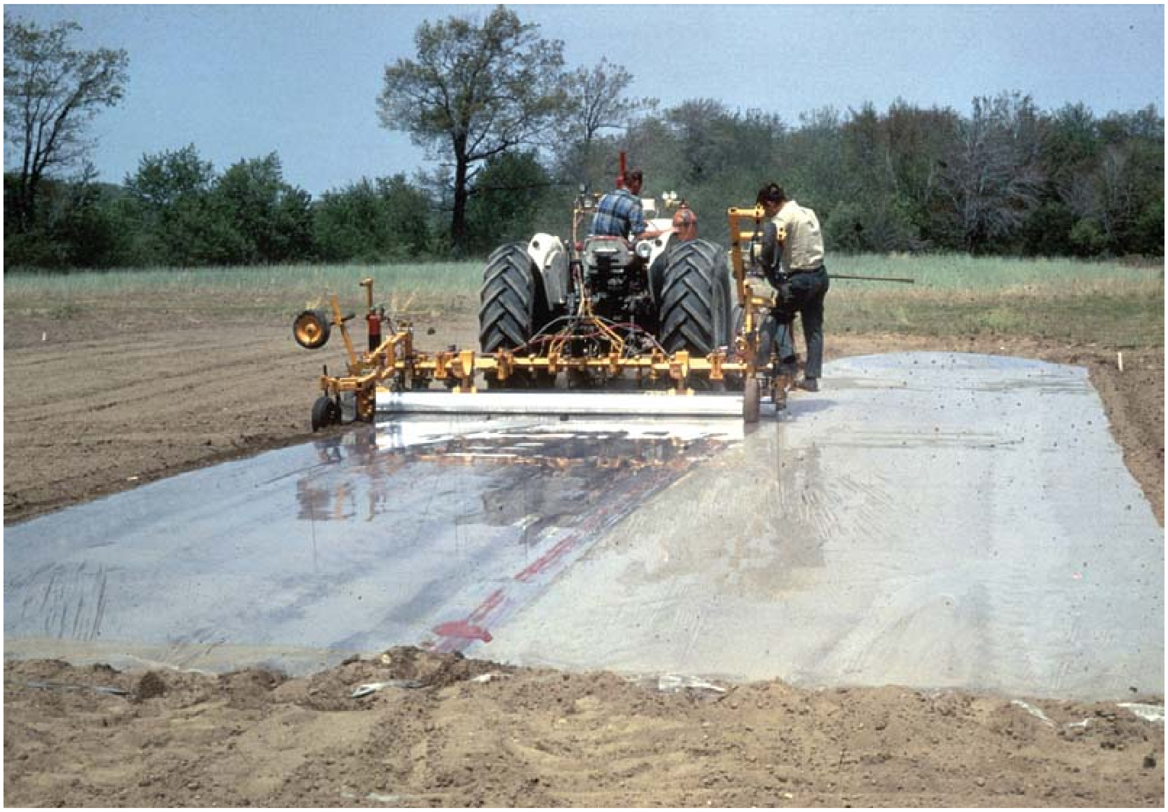


Figure 4.21.
Application of methyl bromide, a soil fumigant, under plastic. Noncritical uses of methyl bromide were phased out in the United States in 2005 because it is considered a threat to the ozone layer. Certain allowances continue to be made for uses for which no technically and economically feasible alternatives have been identified.

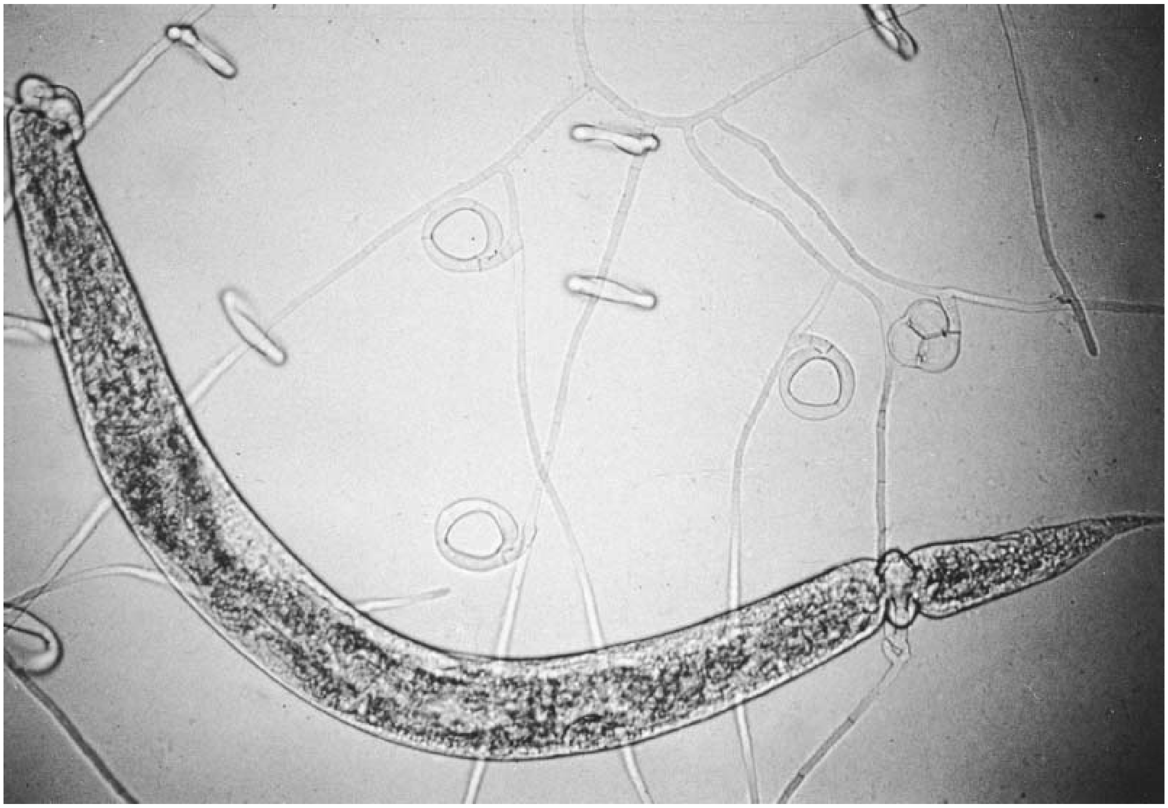


Figure 4.22.
The fungus *Dactylaria brochopaga* traps nematodes by constricting a hyphal noose. It is one of many potential biological control agents for nematodes.

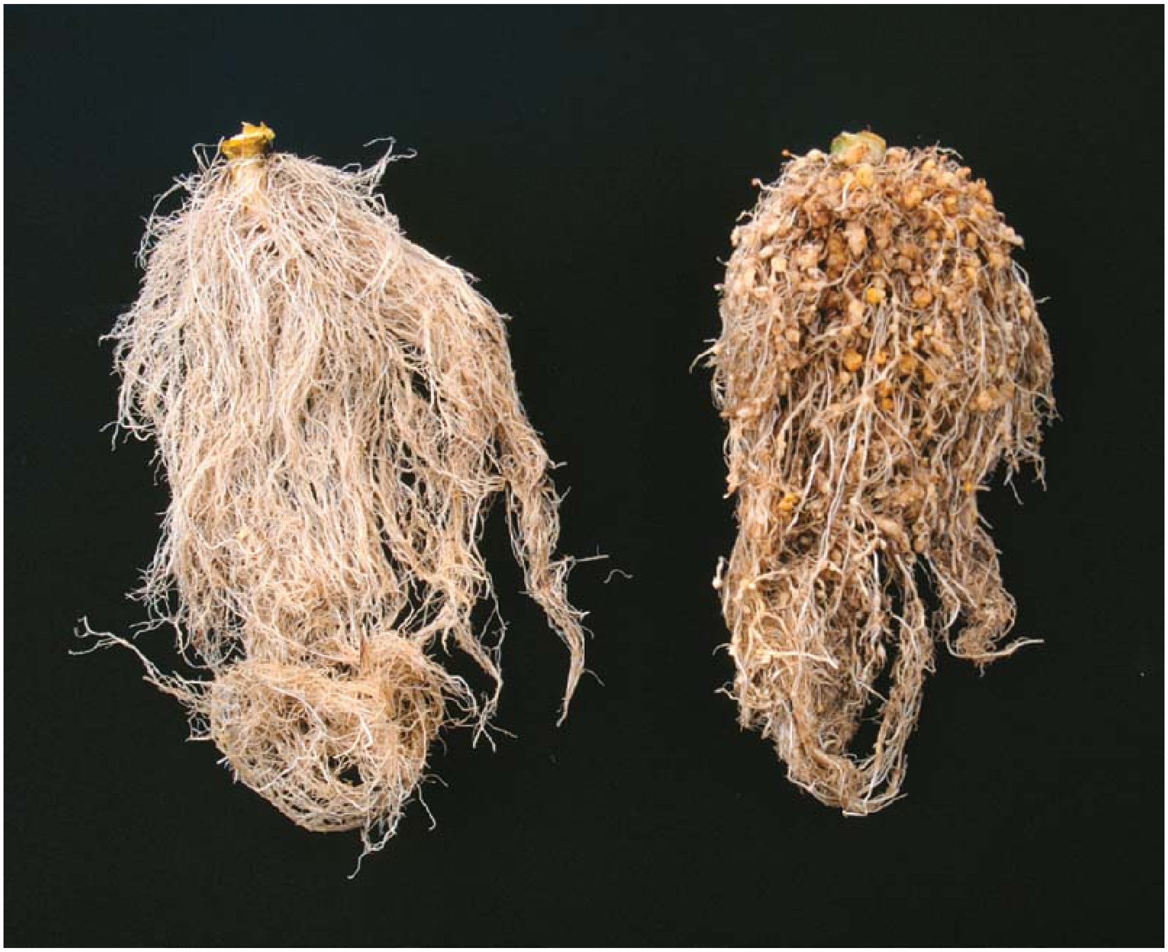


Figure 4.23.
Roots of lettuce cultivar Salinas (left), resistant to root-knot nematodes, and of susceptible lettuce cultivar Ithaca (right).

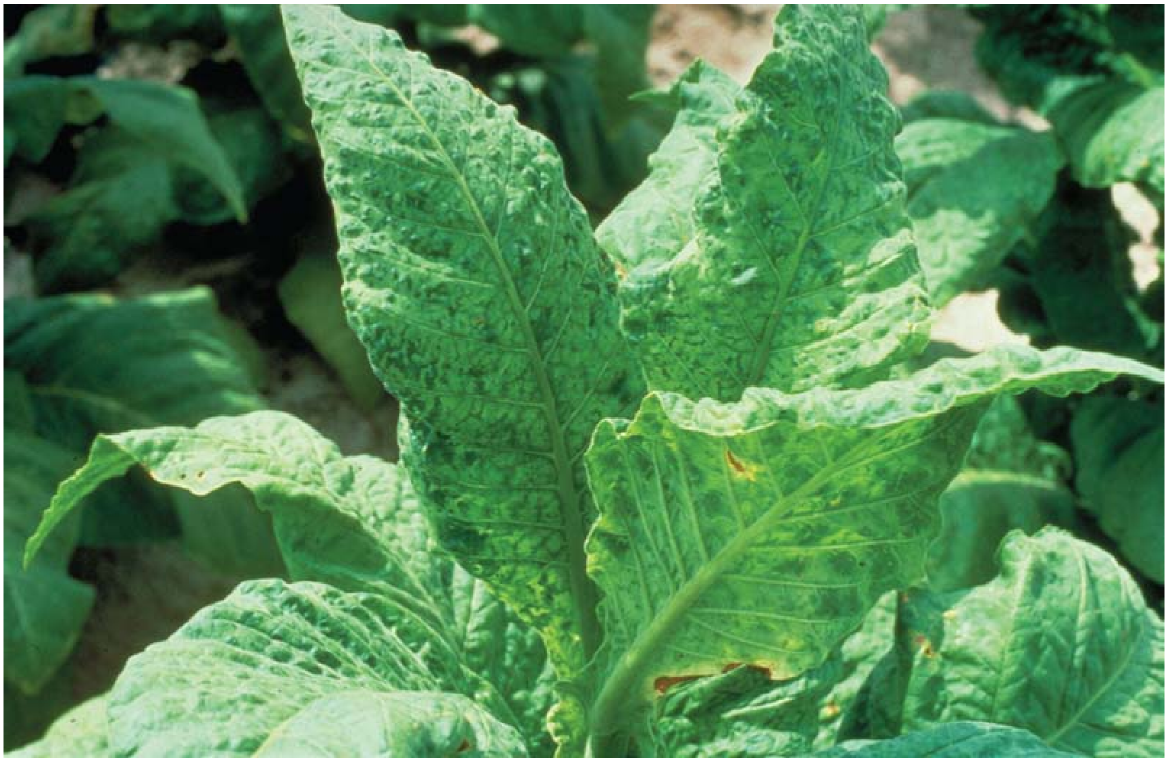


Figure 5.1.
Tobacco leaves with symptoms caused by *Tobacco mosaic virus* (TMV). Scientific studies of TMV began in the 1890s, and the virus has been used since then as a model in the study of viruses and their interactions with host plants.

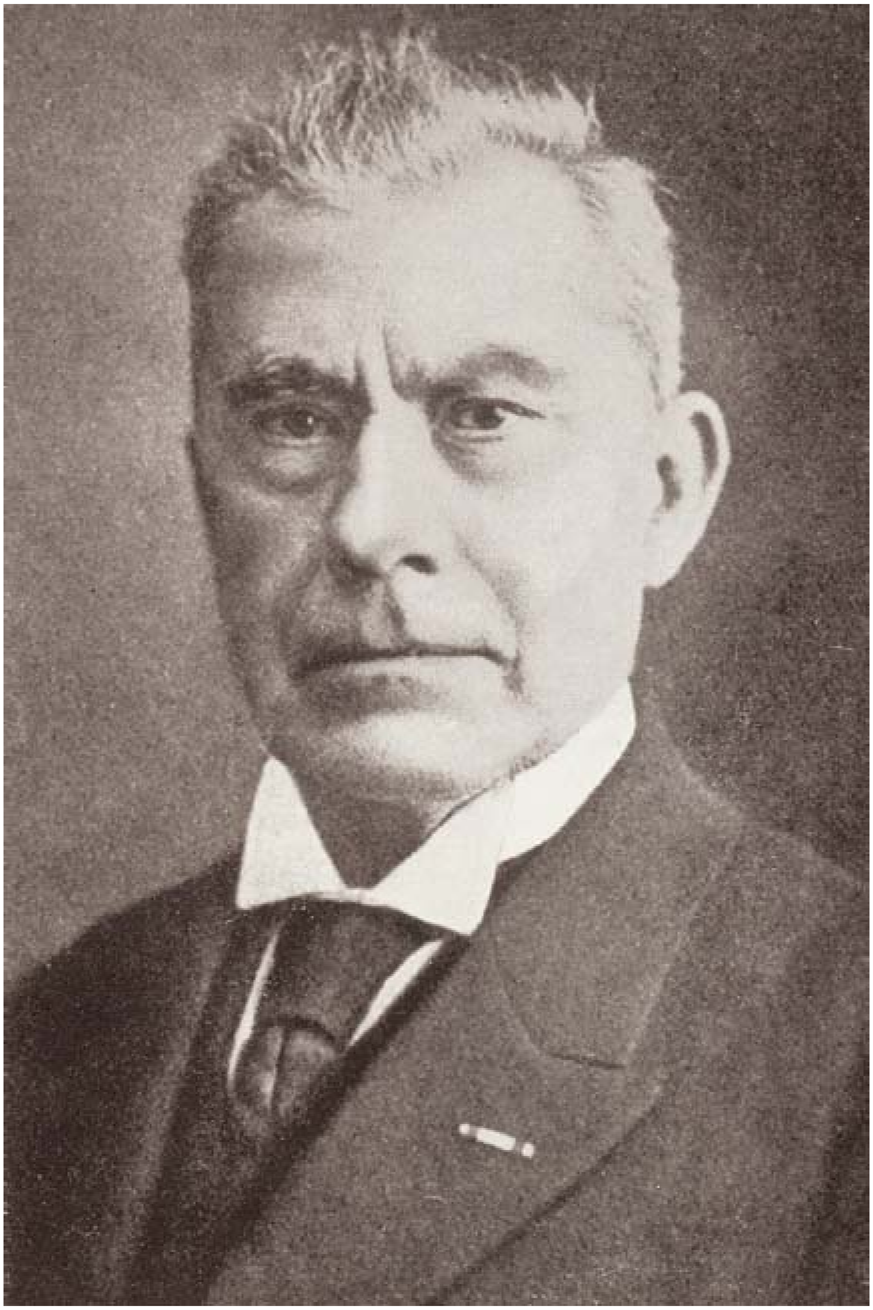


Figure 5.2.
Martinus Willem Beijerinck (1851-1931) was the first scientist to propose that what we now call viruses are fundamentally different from other disease-causing agents, principally bacteria. His concept of a *“contagium vivum fluidum”* (a contagious living fluid) was groundbreaking, and for this insight he has been called the “father of virology.”

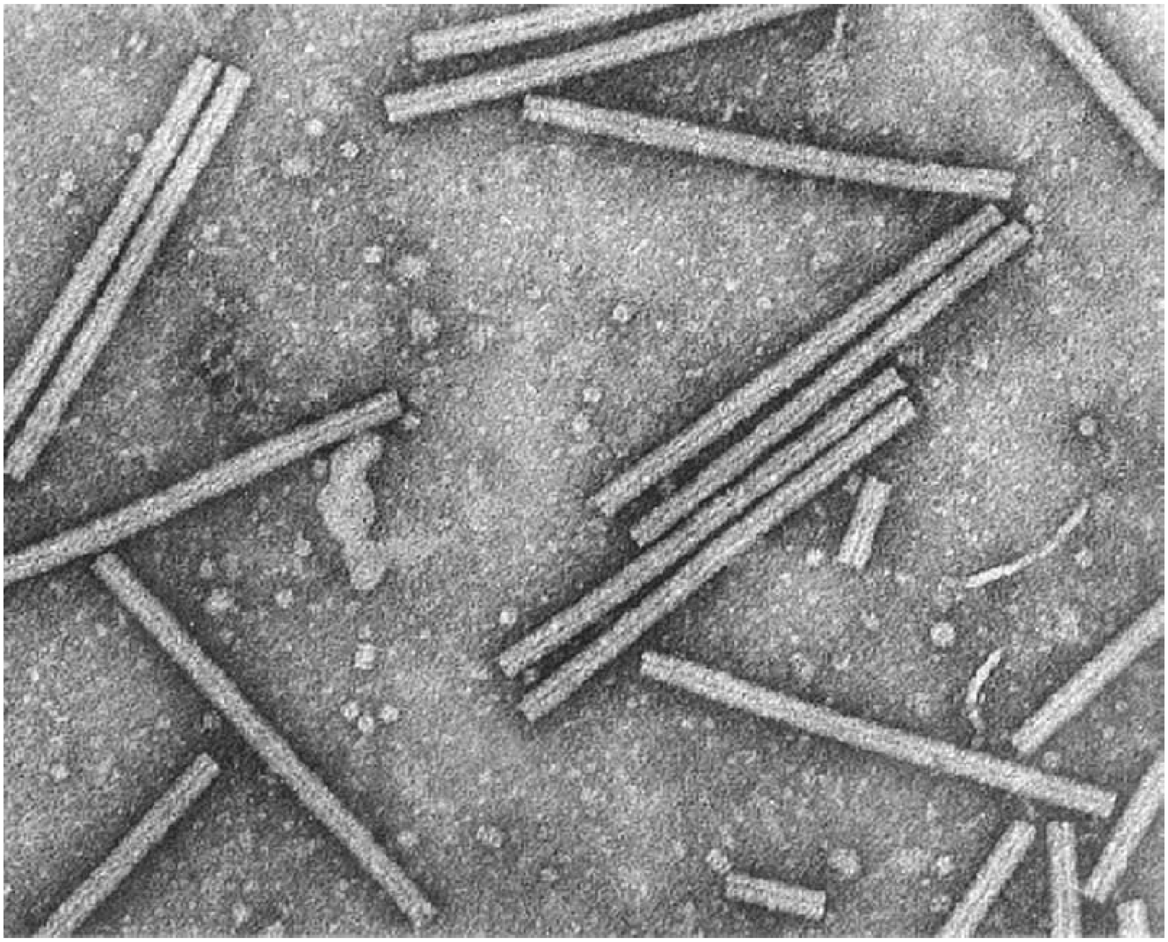


Figure 5.3.
Rod-shaped particles (300 × 15 nm) of *Tobacco mosaic virus*, in an electron micrograph.

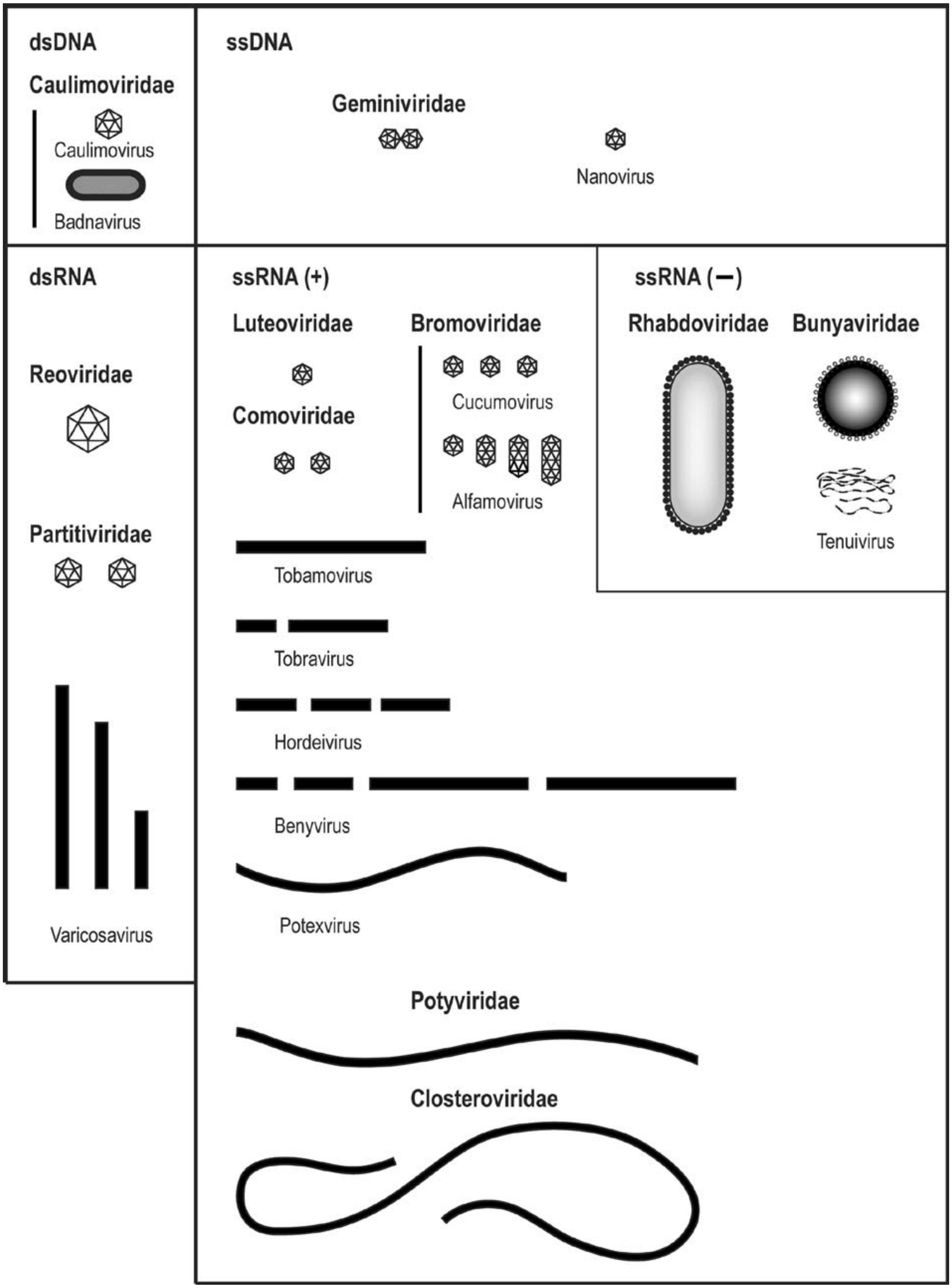

dsDNA
Caulimoviridae
Caulimovirus
Badnavirus
ssDNA
Geminiviridae
Nanovirus
dsRNA
Reoviridae
Partitiviridae
Varicosavirus
ssRNA (+)
Luteoviridae
Comoviridae
Bromoviridae
Cucumovirus
Alfamovirus
Tobamovirus
Tobravirus
Hordeivirus
Benyvirus
Potexvirus
Potyviridae
Closteroviridae
ssRNA (−)
Rhabdoviridae
Bunyaviridae
Tenuivirus

Figure 5.4.

Some of the families and genera of plant-pathogenic viruses. Note the variety of shapes of virus particles and the different types of nucleic acid genomes.

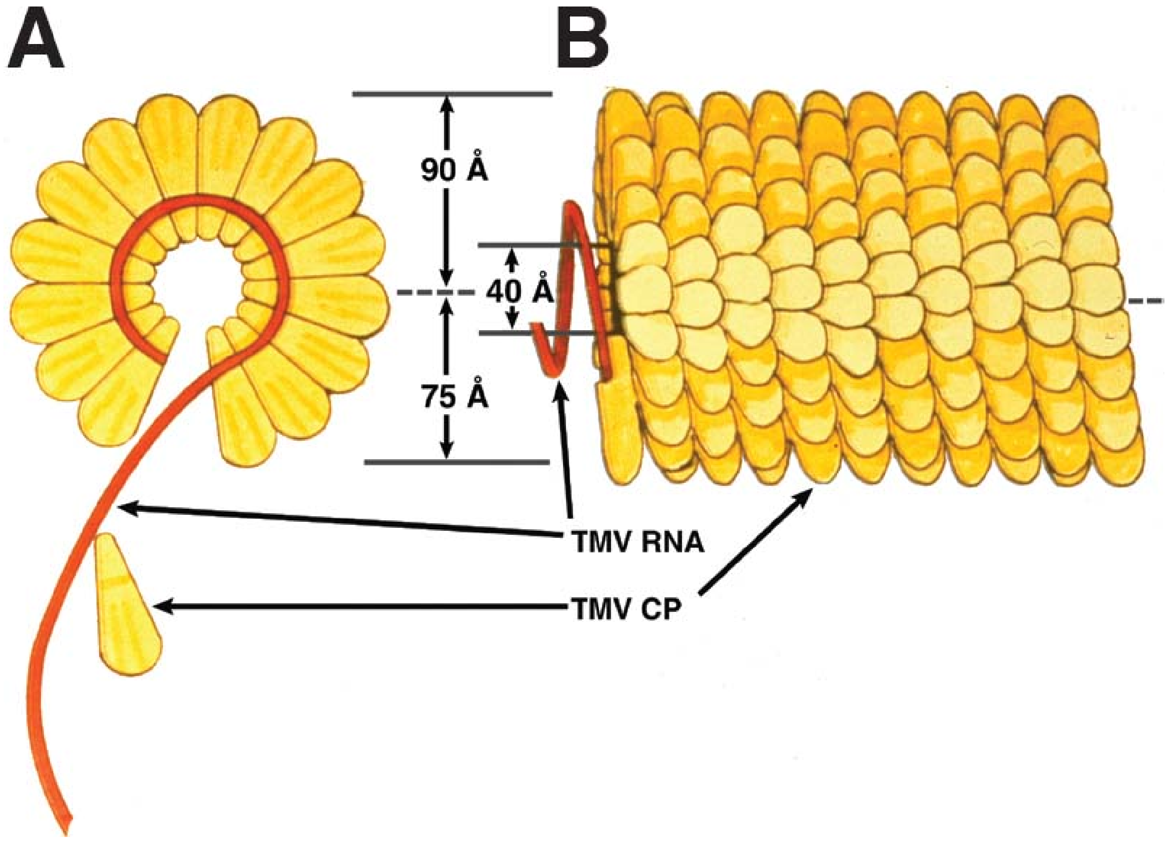


Figure 5.5.
Structure of a *Tobacco mosaic virus* (TMV) particle. The virus particle is made up of a spiral or helix of 2,130 coat protein (CP) subunits and a single-stranded positive-sense RNA of approximately 6,400 nucleotides. The relationship between the CP subunits and the single-stranded RNA is shown in a transverse view (**A**) and a longitudinal view (**B**) (10 Å = 1 nm).

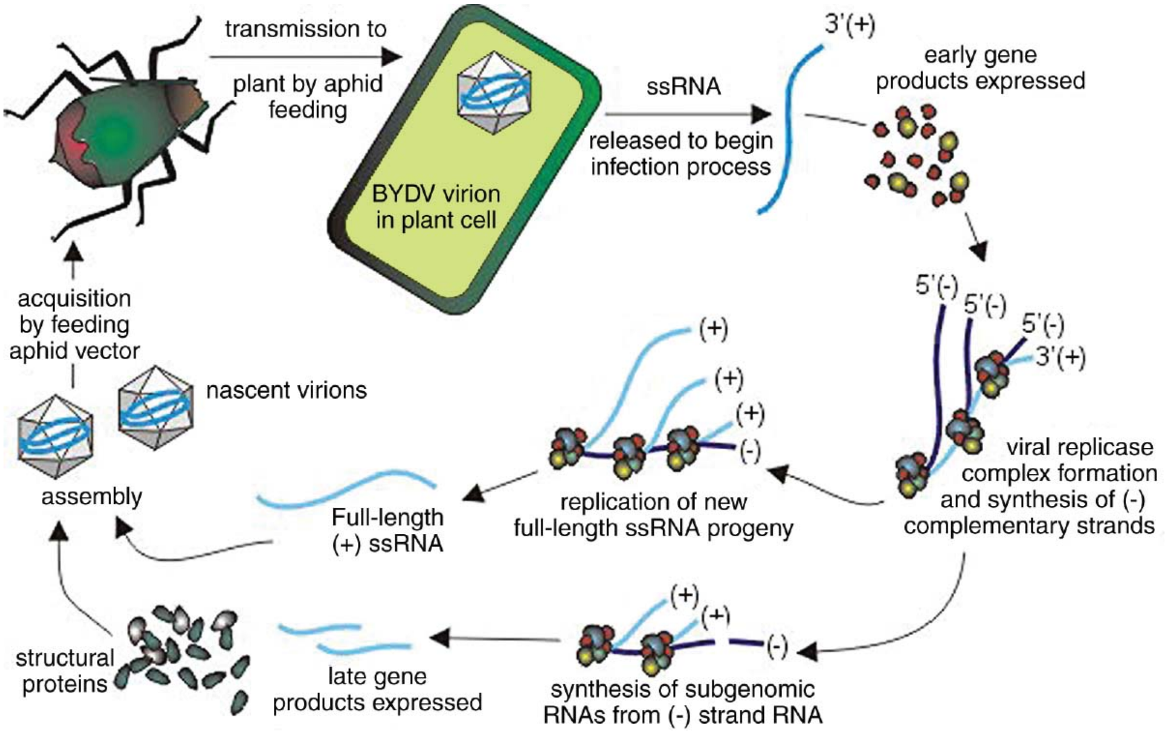

transmission to
plant by aphid feeding
BYDV virion in plant cell
ssRNA
released to begin infection process
3'(+)
early gene products expressed
5'(-)
5'(-)
5'(-)
3'(+)
viral replicase complex formation and synthesis of (-) complementary strands
(+)
(+)
(+)
(-)
replication of new full-length ssRNA progeny
Full-length (+) ssRNA
(+)
(+)
(-)
synthesis of subgenomic RNAs from (-) strand RNA
late gene products expressed
structural proteins
assembly
nascent virions
acquisition by feeding aphid vector

Figure 5.6.

Replication cycle of *Barley yellow dwarf virus* (BYDV), a +sense ssRNA virus.

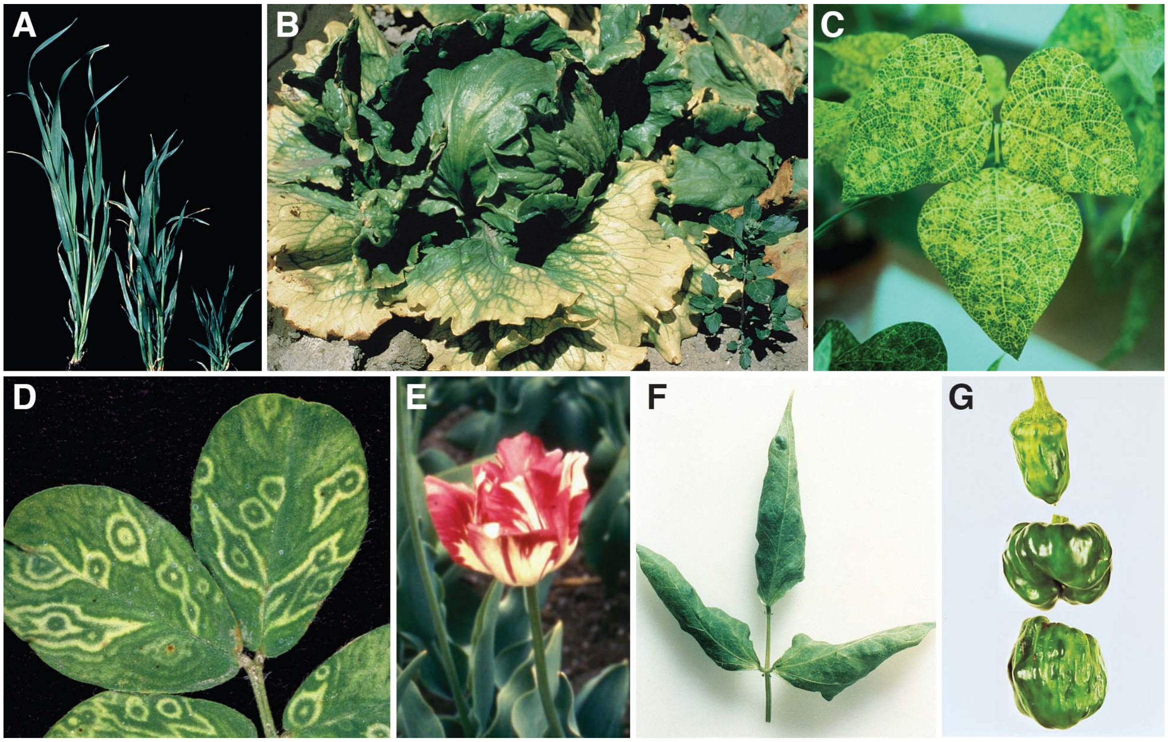


Figure 5.7.

Common symptoms caused by viruses. **A**, Stunting or dwarfing (a healthy plant is on the left). **B**, Yellows. **C**, Mosaic. **D**, Ringspot. **E**, Flower break. **F**, Leafroll. **G**, Fruit deformation.

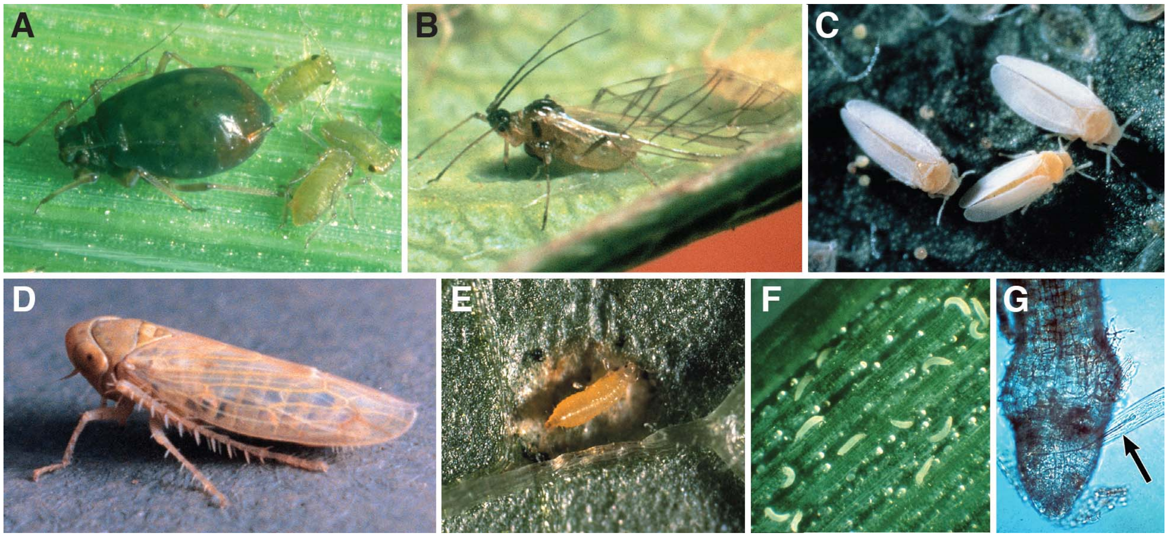


Figure 5.8.
Vectors of plant viruses. **A,** Wingless (apterous) aphids. **B,** Winged (alate) aphid. **C,** Whiteflies. **D,** Leafhopper. **E,** Thrips (larva). **F,** Eriophyid mites. **G,** Migratory ectoparasitic nematode (arrow) feeding at a root tip.

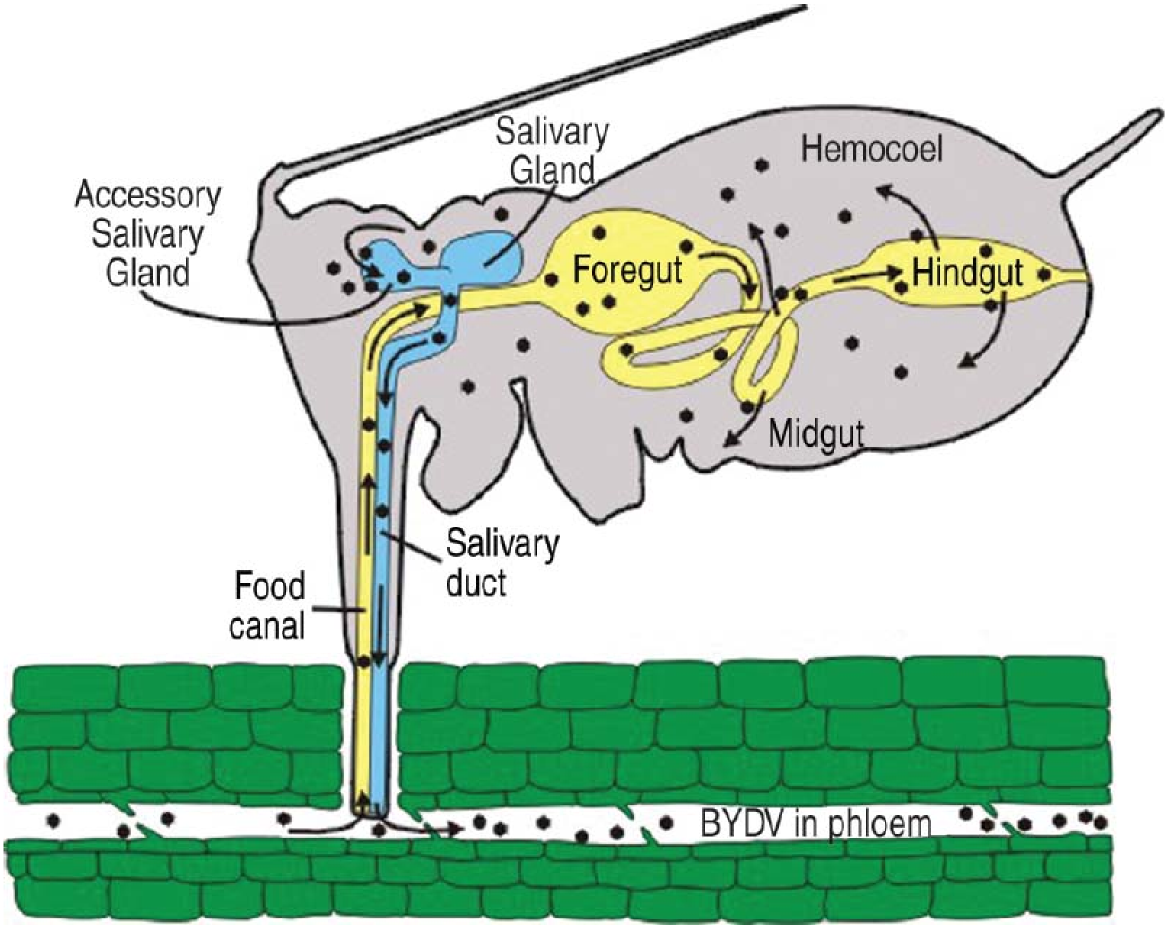

Salivary Gland
Hemocoel
Accessory Salivary Gland
Foregut
Hindgut
Midgut
Salivary duct
Food canal
BYDV in phloem

Figure 5.9.
Aphid vector feeding on a plant host and the internal route of the viruses that cause barley yellow dwarf. These viruses are transmitted in a persistent (circulative) manner.

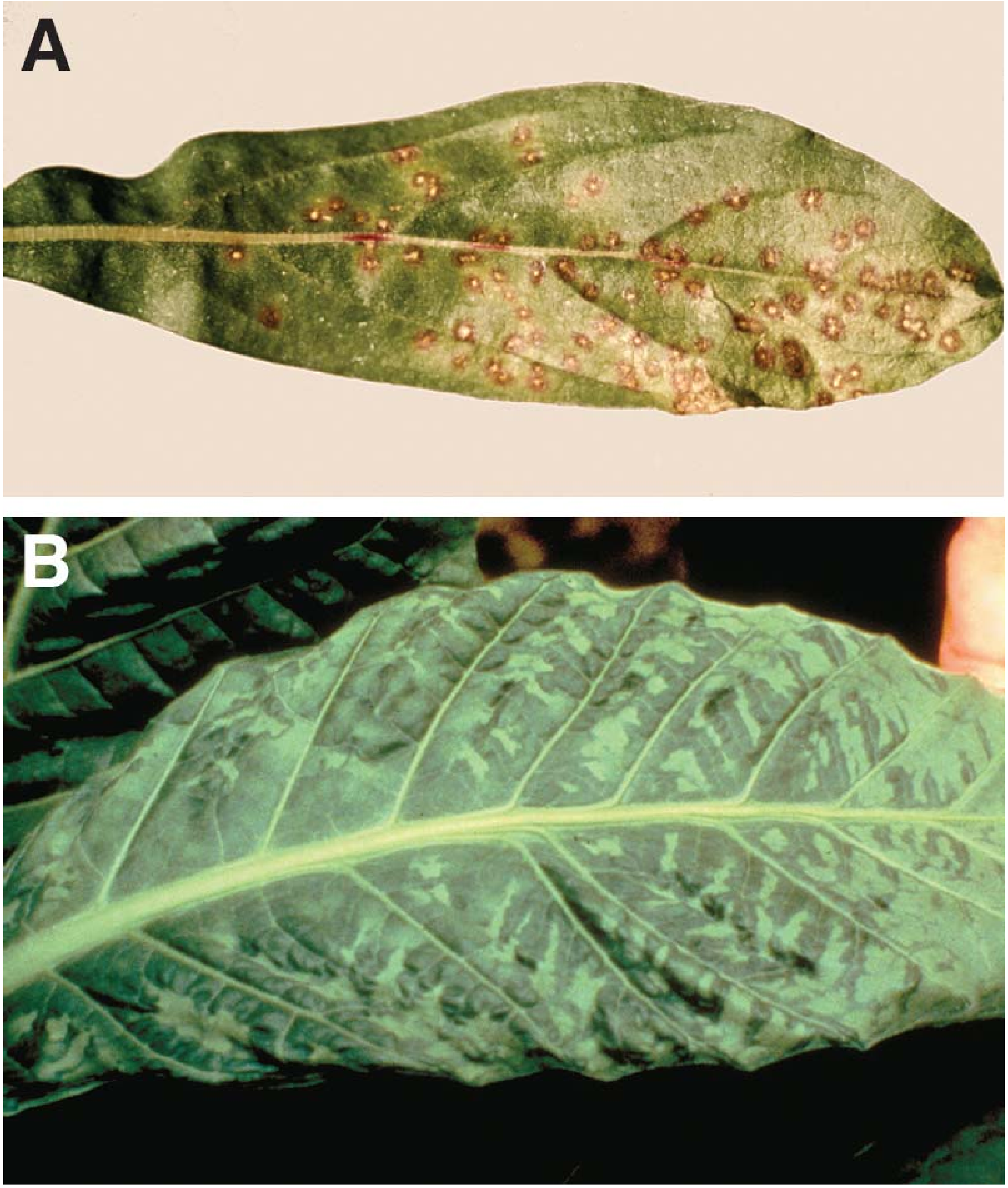


Figure 5.10.
Local lesions (**A**) and systemic symptoms (**B**) resulting from virus inoculations.

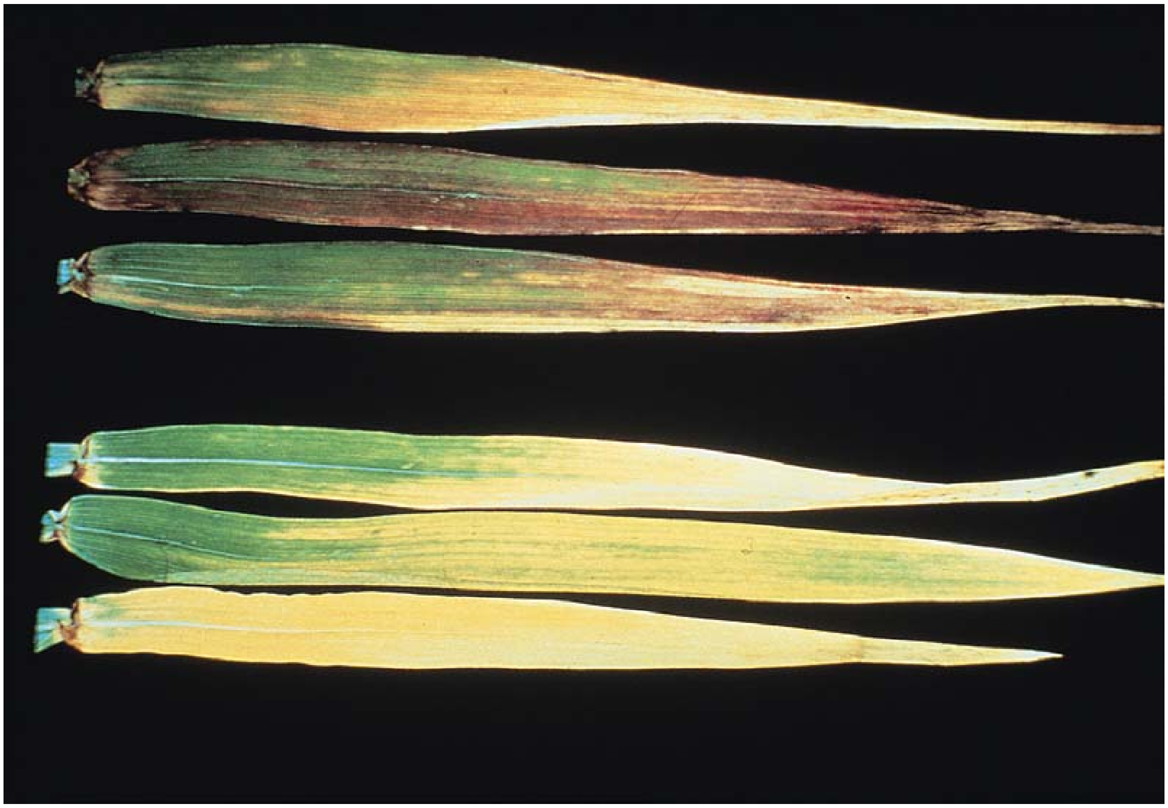


Figure 5.11.
Leaf discoloration caused by *Barley yellow dwarf virus*.

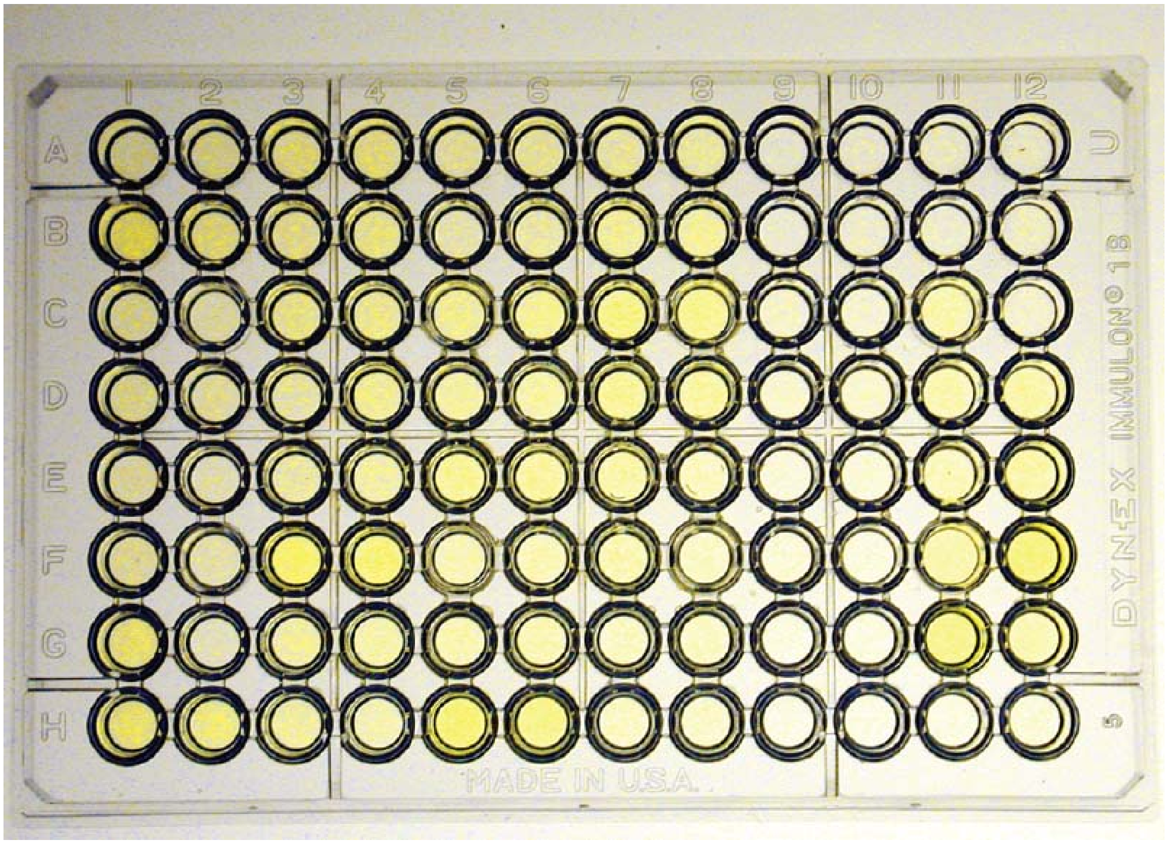

1
2
3
4
5
6
7
8
9
10
11
12
A
B
C
D
E
F
G
H
DYNEX IMMULON® 1B
MADE IN U.S.A.

Figure 5.12.
Microtiter plate used for enzyme-linked immunosorbent assay (ELISA). Wells containing virus develop color; wells without virus do not.

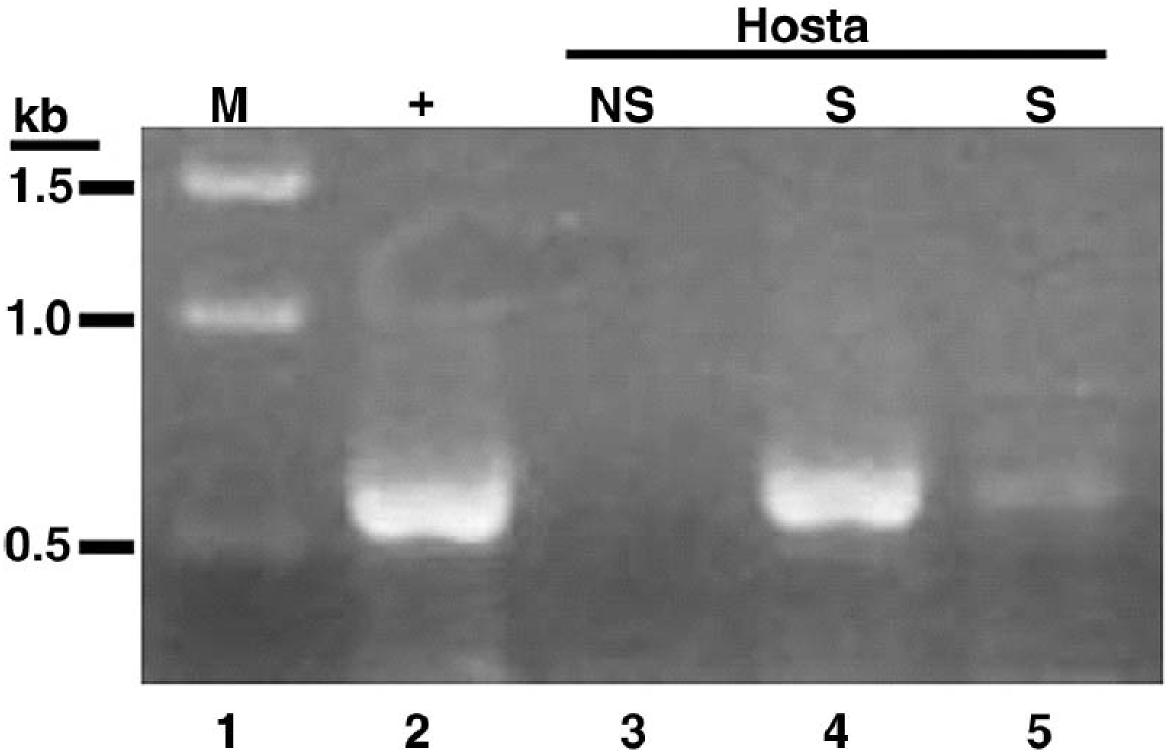

Hosta
kb
M
+
NS
S
S
1.5
1.0
0.5
1
2
3
4
5

Figure 5.13.
Detection of *Tomato spotted wilt virus* infection in hosta by reverse transcription polymerase chain reaction (RT-PCR).

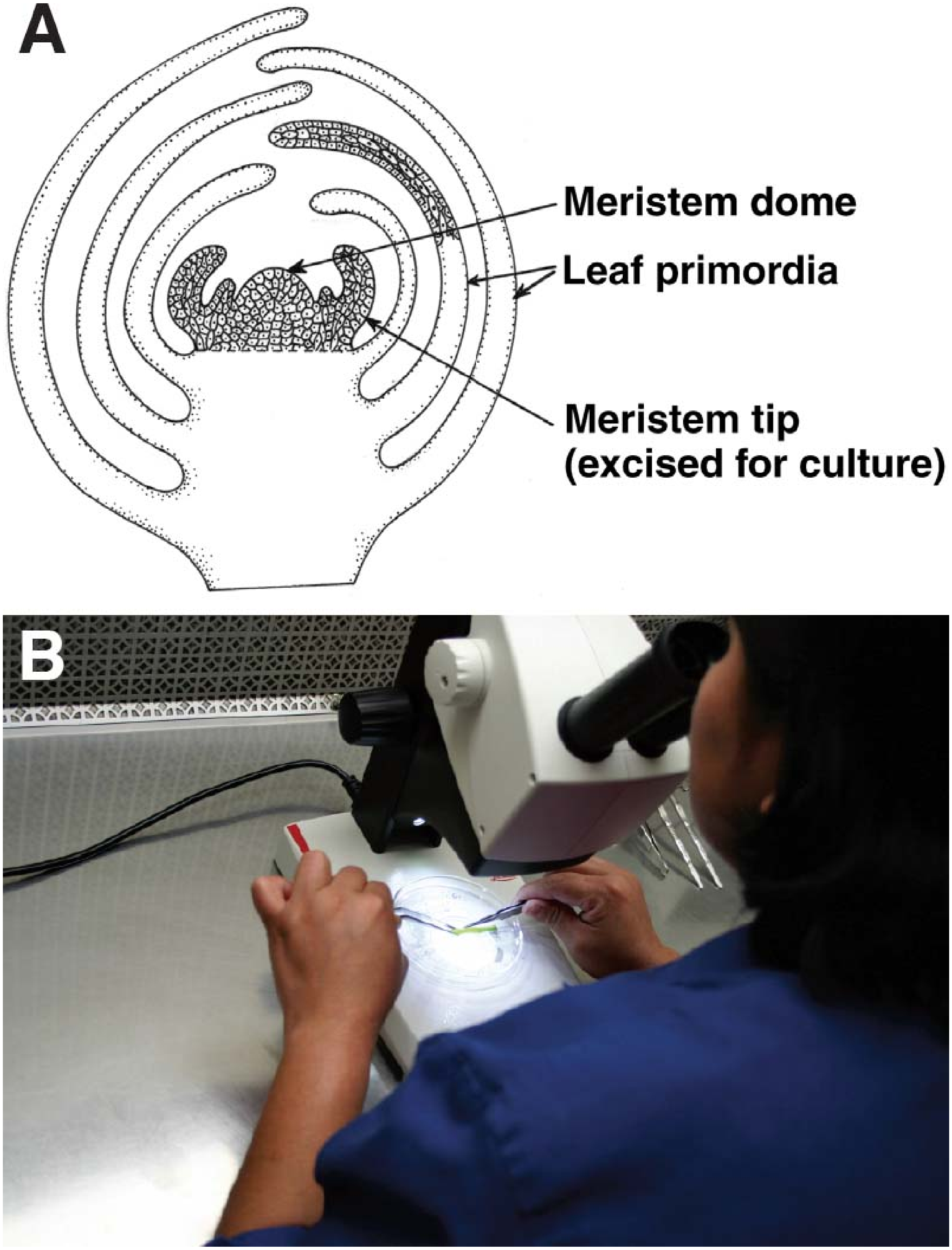

A
Meristem dome
Leaf primordia
Meristem tip
(excised for culture)
B

Figure 5.14.
Meristem tip culture. **A,** Cross section of a bud, showing the meristem tip region (above the dotted line). **B,** Removal of a meristem tip with

the use of a dissecting microscope.

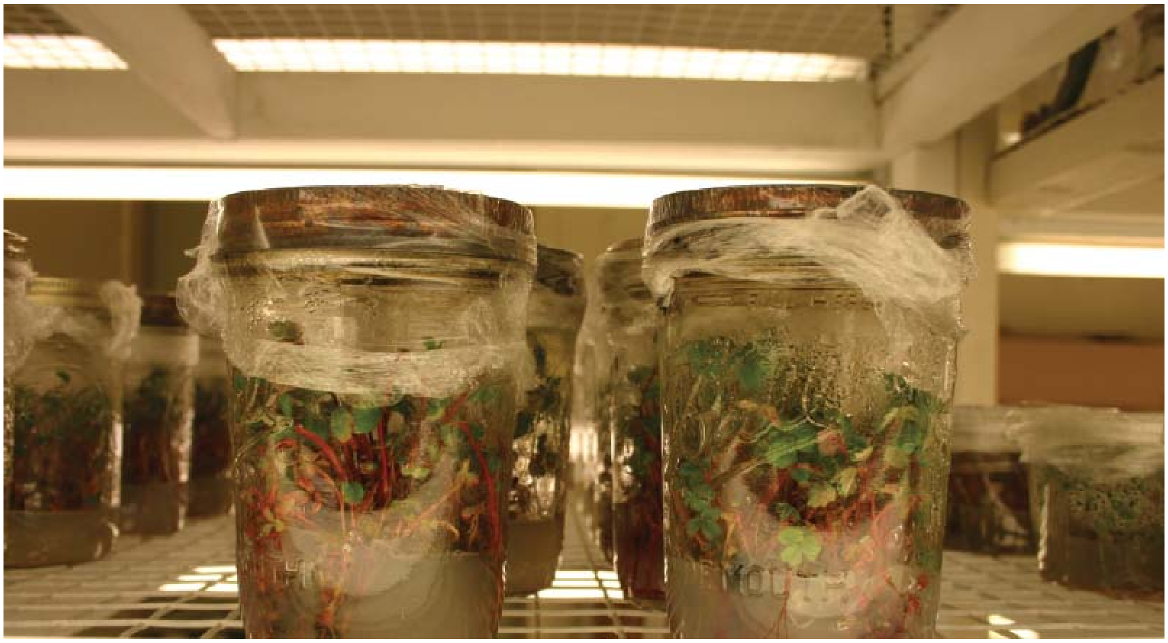


Figure 5.15.
Tissue culture jars in a laboratory where thousands of plants are propagated in a virus-free environment.

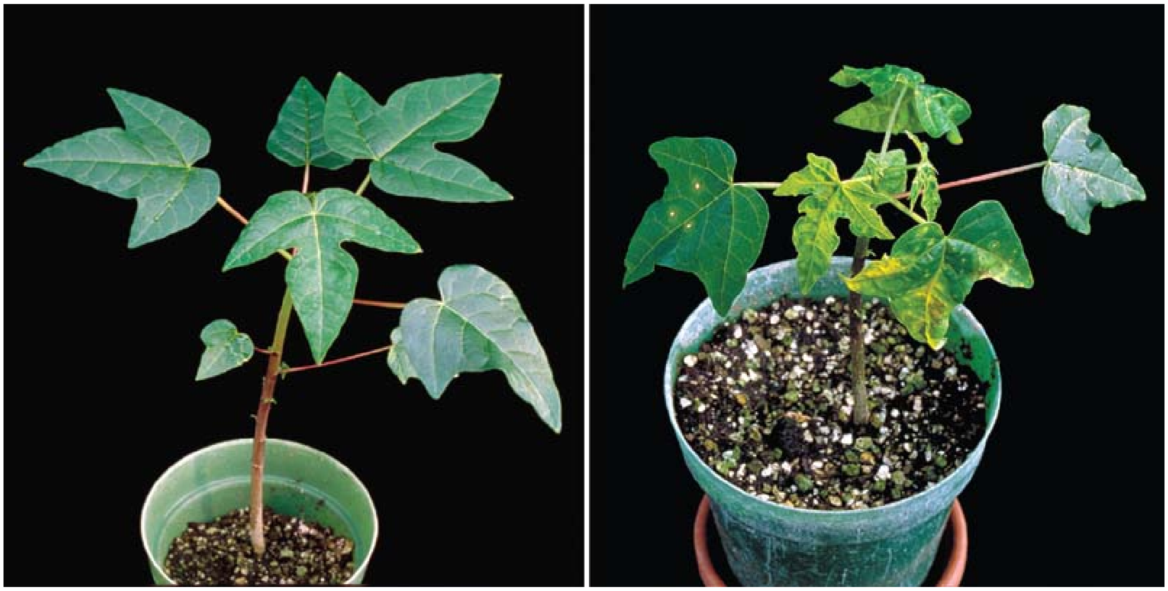


Figure 5.16.

Transgenic papaya (left) and nontransgenic papaya (right) inoculated with *Papaya ringspot virus*. The transgenic papaya contains the gene for the viral coat protein, which confers protection from the virus.

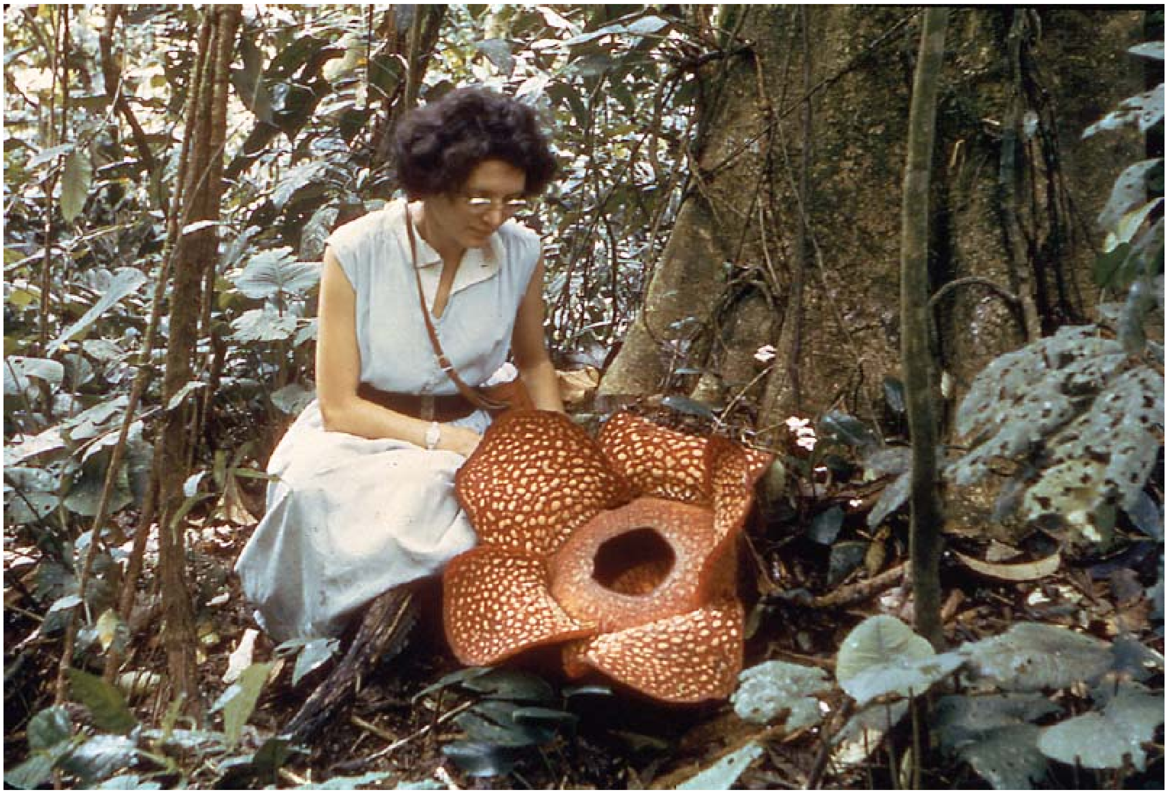


Figure 6.1.
Rafflesia arnoldii, a holoparasitic plant.

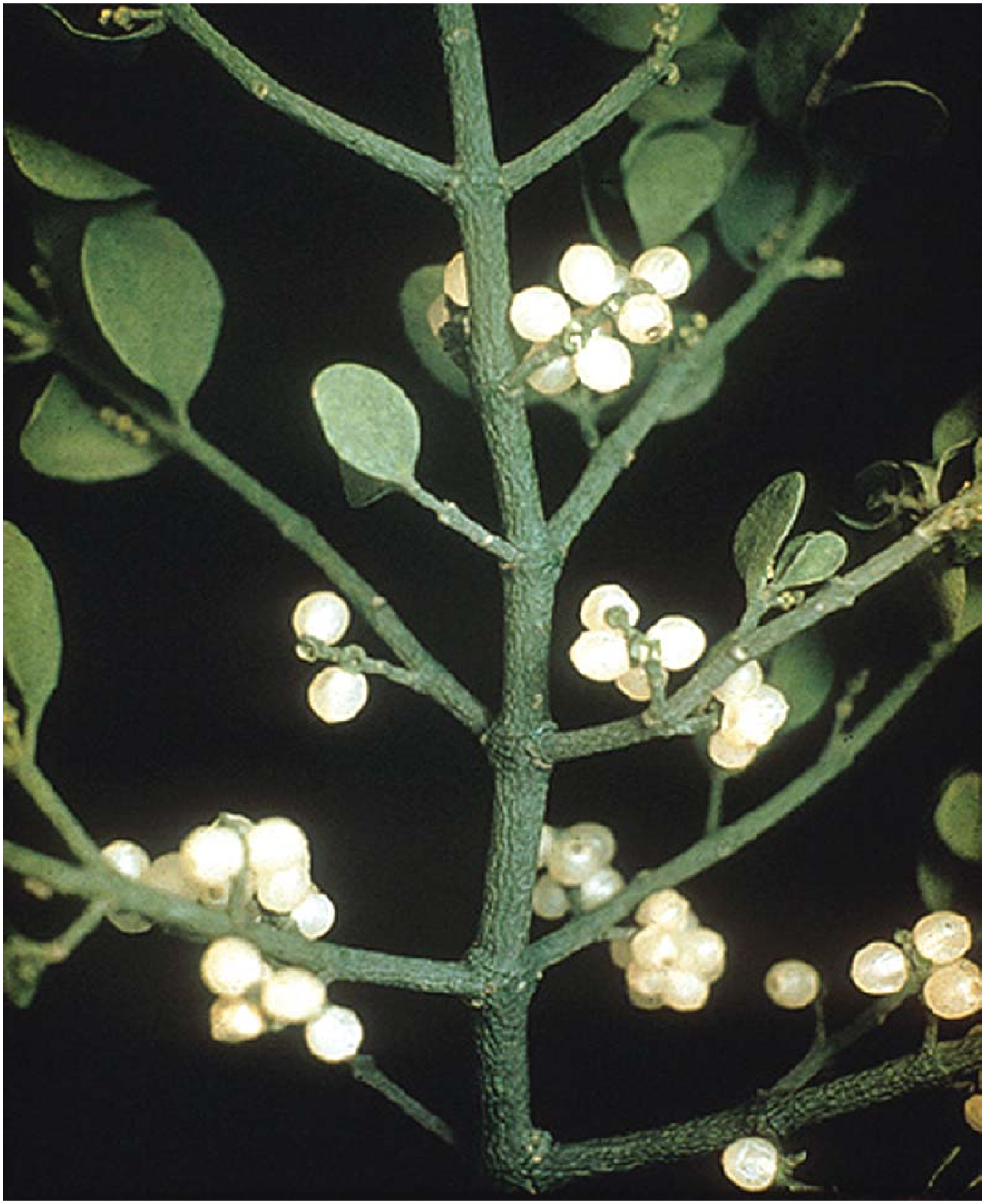


Figure 6.2.
Leaves, stems, and berries of mistletoe (*Phoradendron serotinum*).

Figure 6.3.
Ponderosa pine (*Pinus ponderosa* var. *scopulorum*) with a witches' broom caused by dwarf mistletoe (*Arceuthobium vaginatum* ssp. *cryptopodum*).

Figure 6.4.
Broomrape (*Orobanche ramosa*) parasitizing tomato.

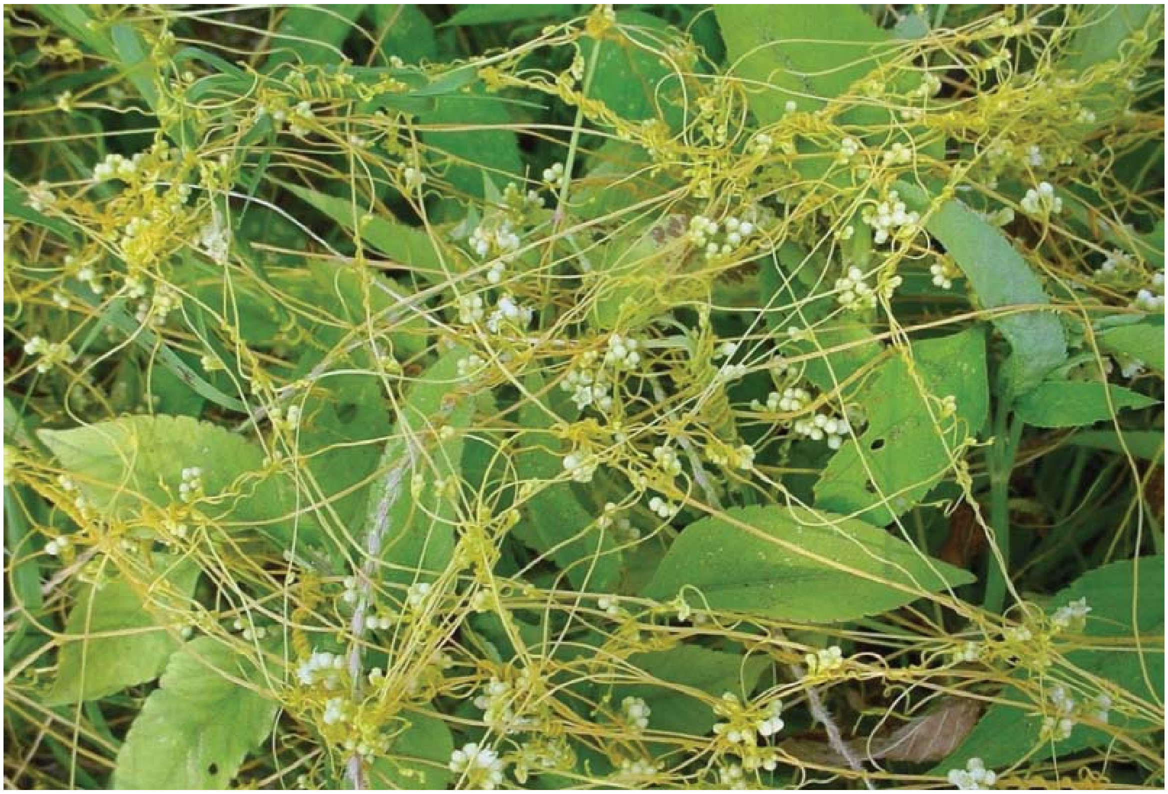


Figure 6.5.
Dodder (*Cuscuta pentagona*) parasitizing herbaceous plants.

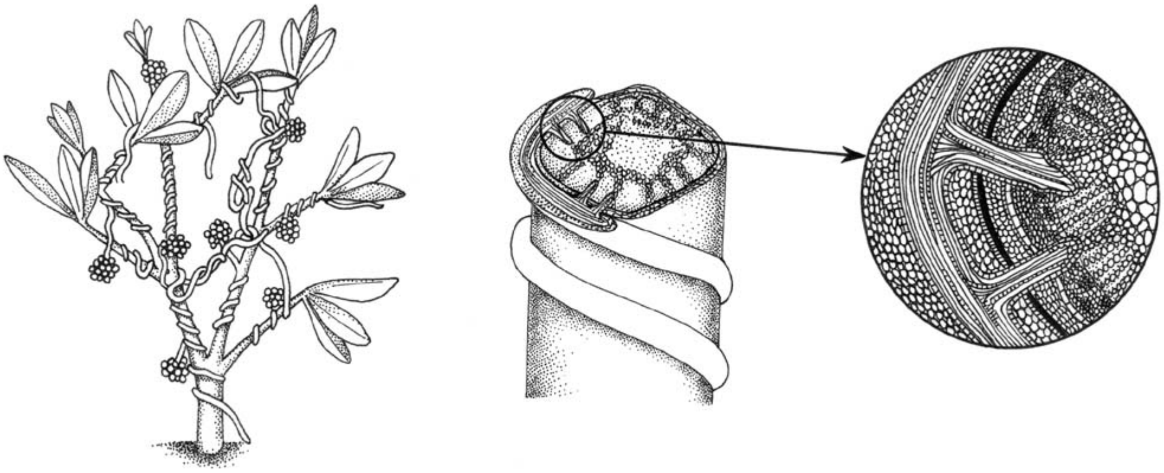


Figure 6.6.
Dodder parasitizing a host plant (left). The invading haustoria (center and right) absorb water and nutrients from the phloem and xylem of the host.

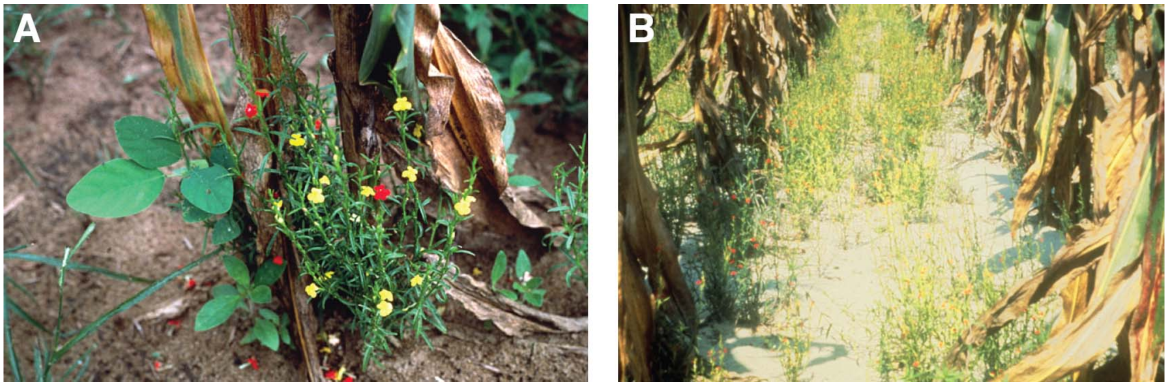


Figure 6.7.
A, Witchweed (*Striga* sp.) parasitizing the roots of a corn plant. **B,** Corn growing poorly because of witchweed infection. Note the witchweed plants between the corn rows.

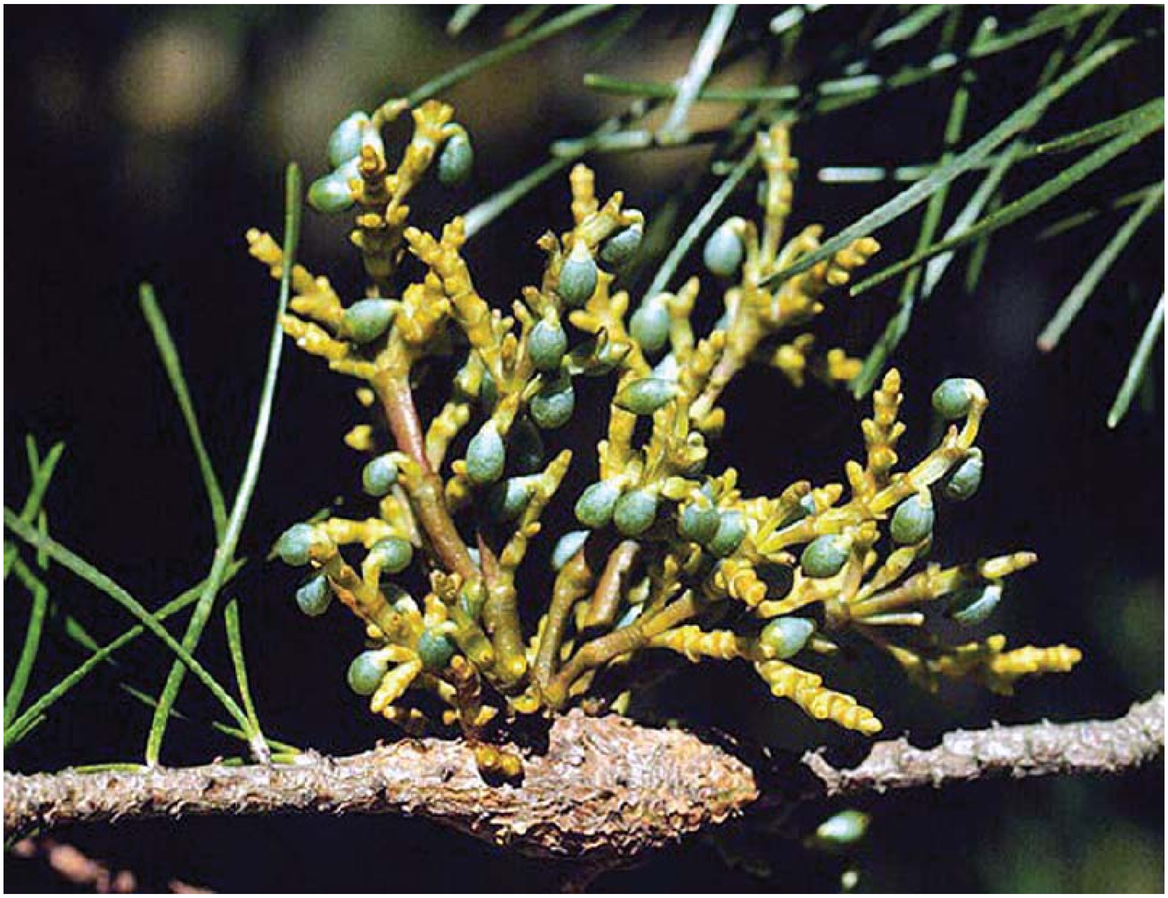


Figure 6.8.
A female (fruiting) dwarf mistletoe (*Arceuthobium campylopodum*) parasitizing a shore pine in British Columbia, Canada.

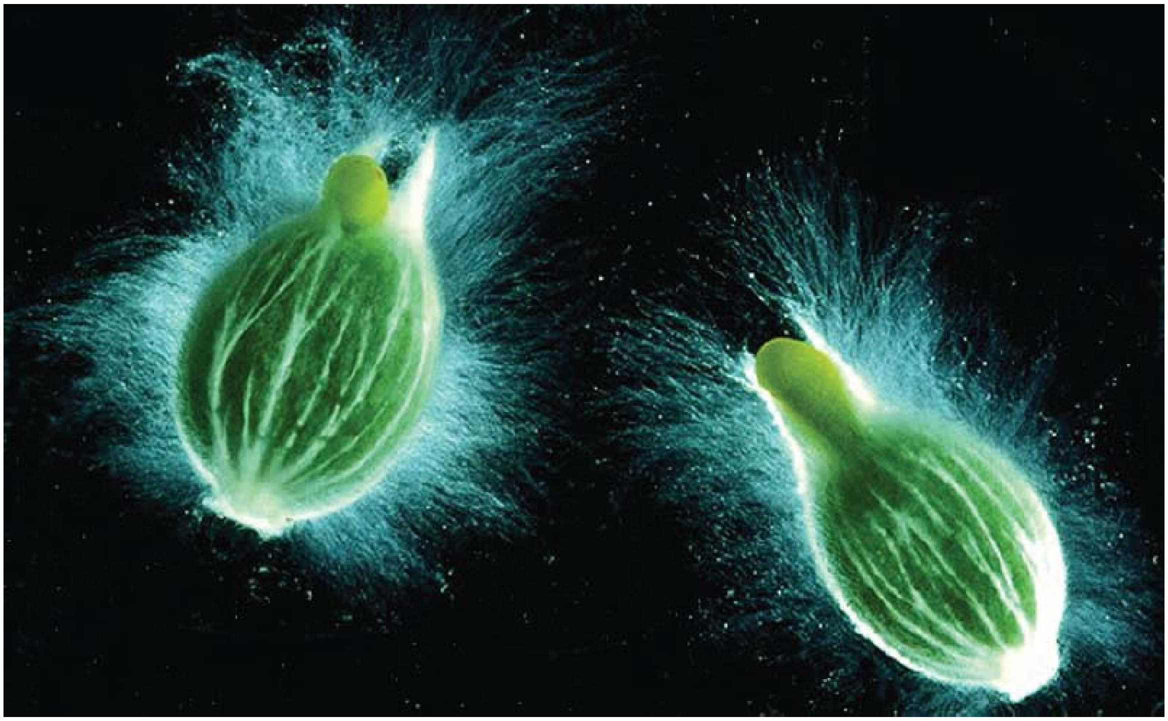


Figure 6.9.
Seeds of a leafy mistletoe (*Phoradendron serotinum*) floating in water. Note that the viscin coats are fully expanded and their cellulosic fibrils are visible.

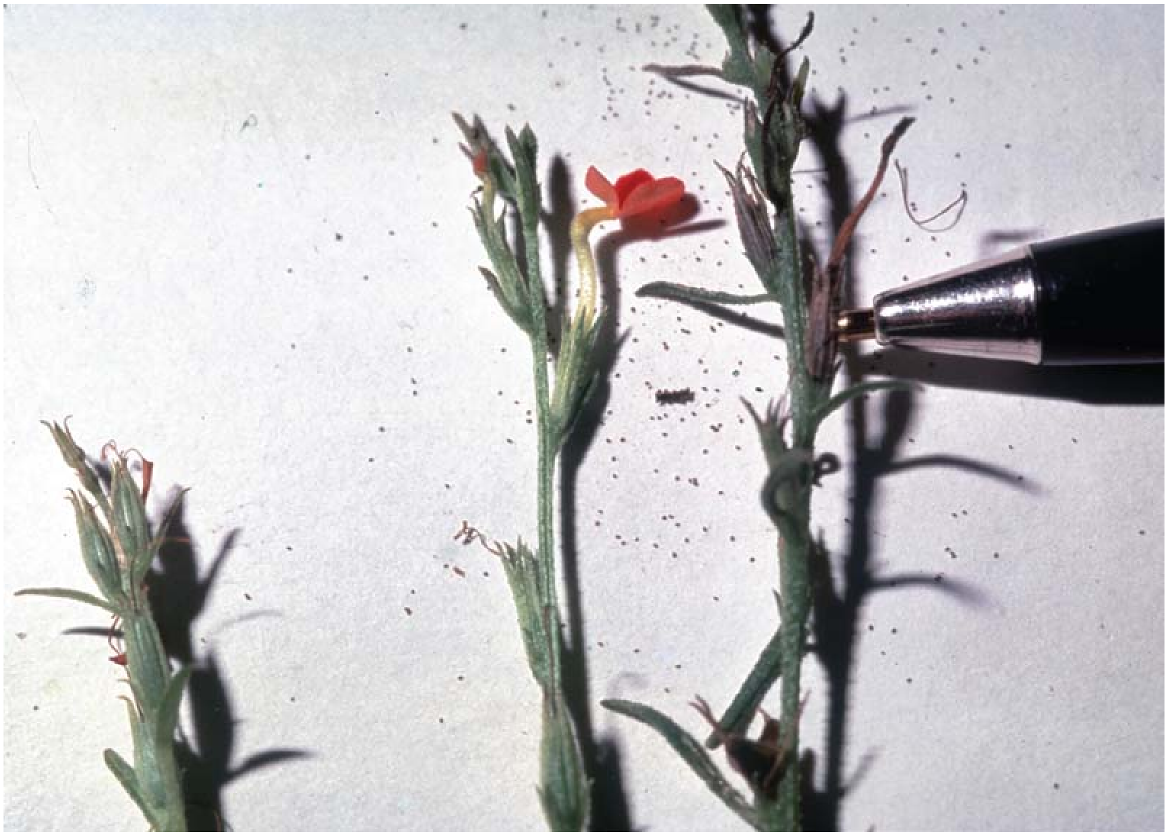


Figure 6.10.
Witchweed (*Striga* sp.) seeds, scattered on the paper beneath the plants, are numerous and tiny.

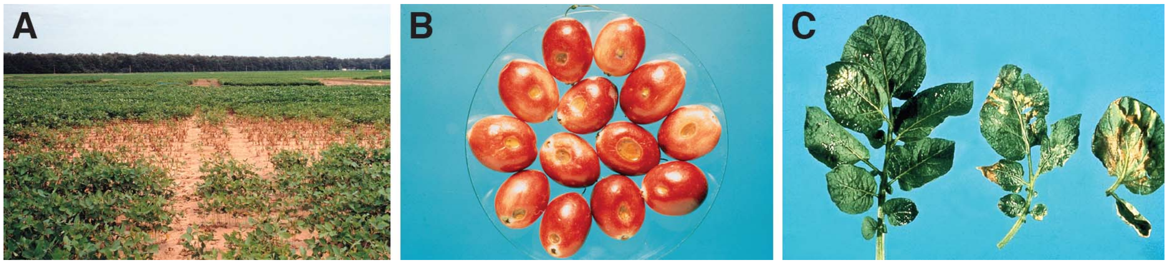


Figure 7.1.
Injury caused by **A,** lightning in a soybean field; **B,** hail on cranberries; and **C,** wind in potato plants.

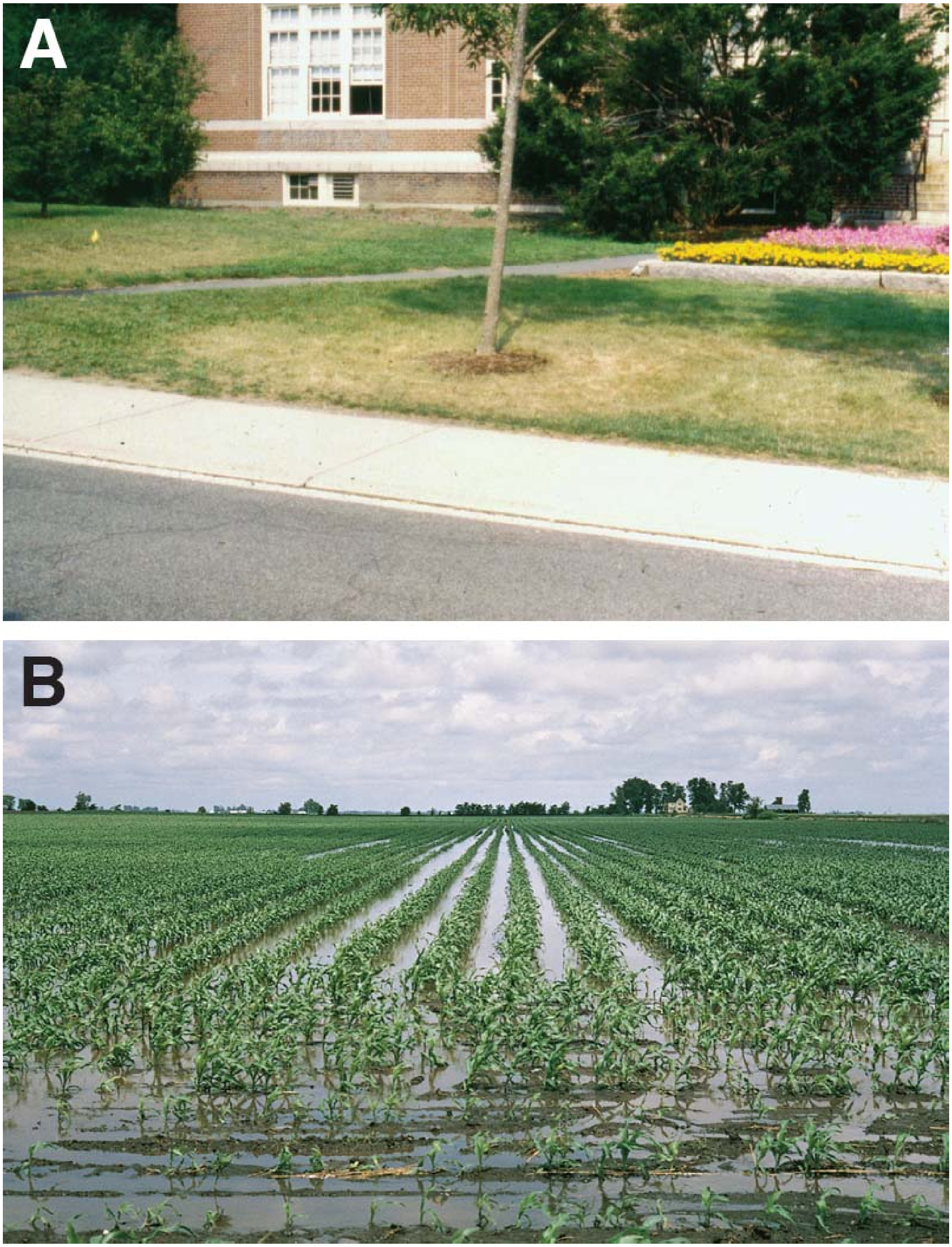


Figure 7.2.
A, Drought stress symptoms on turfgrass. **B,** Flooding injury on corn.

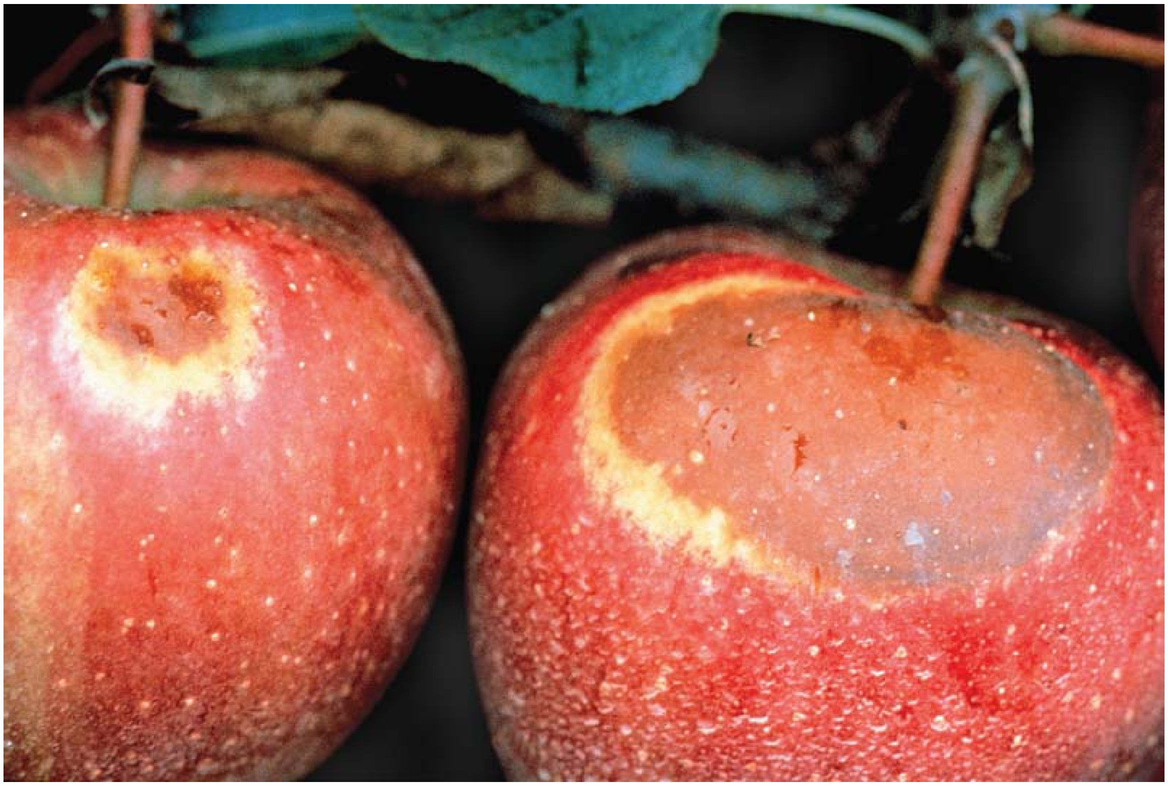


Figure 7.3.
Sunburn on Delicious apples.

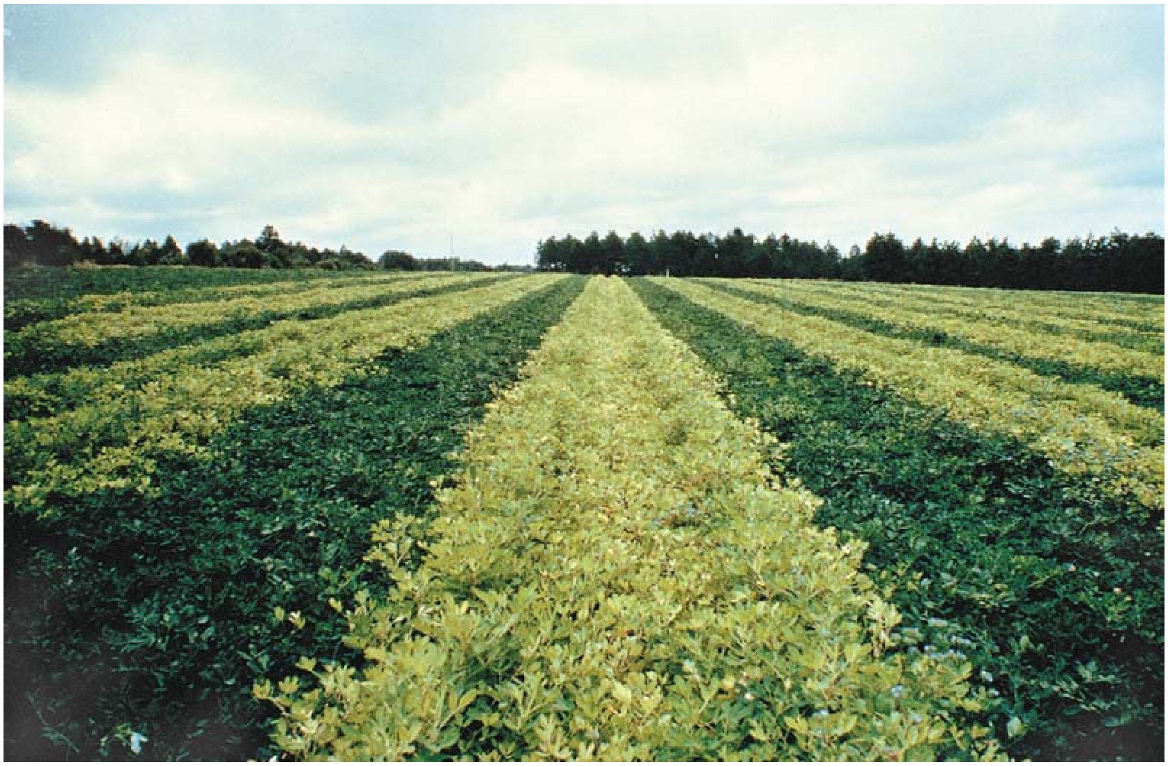


Figure 7.4.
Nitrogen deficiency symptoms on peanut.

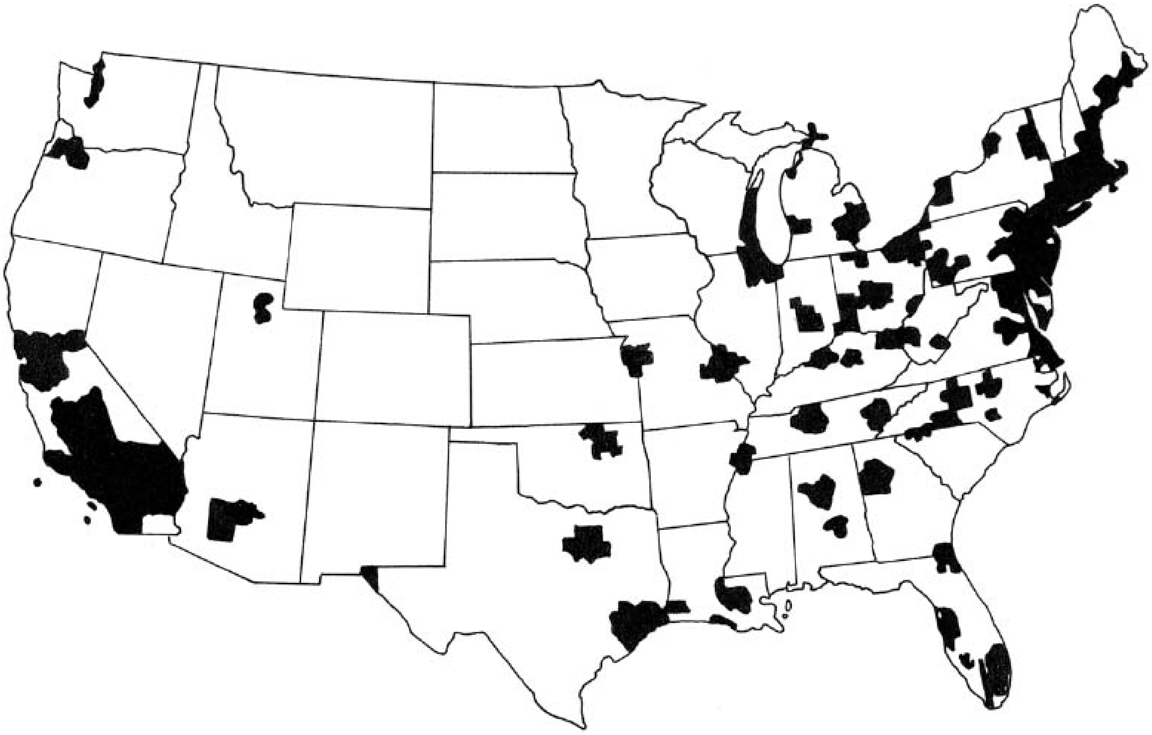


Figure 7.5.
U.S. regions that violated federal air-quality standards for ozone more than once a year for three years in a row.

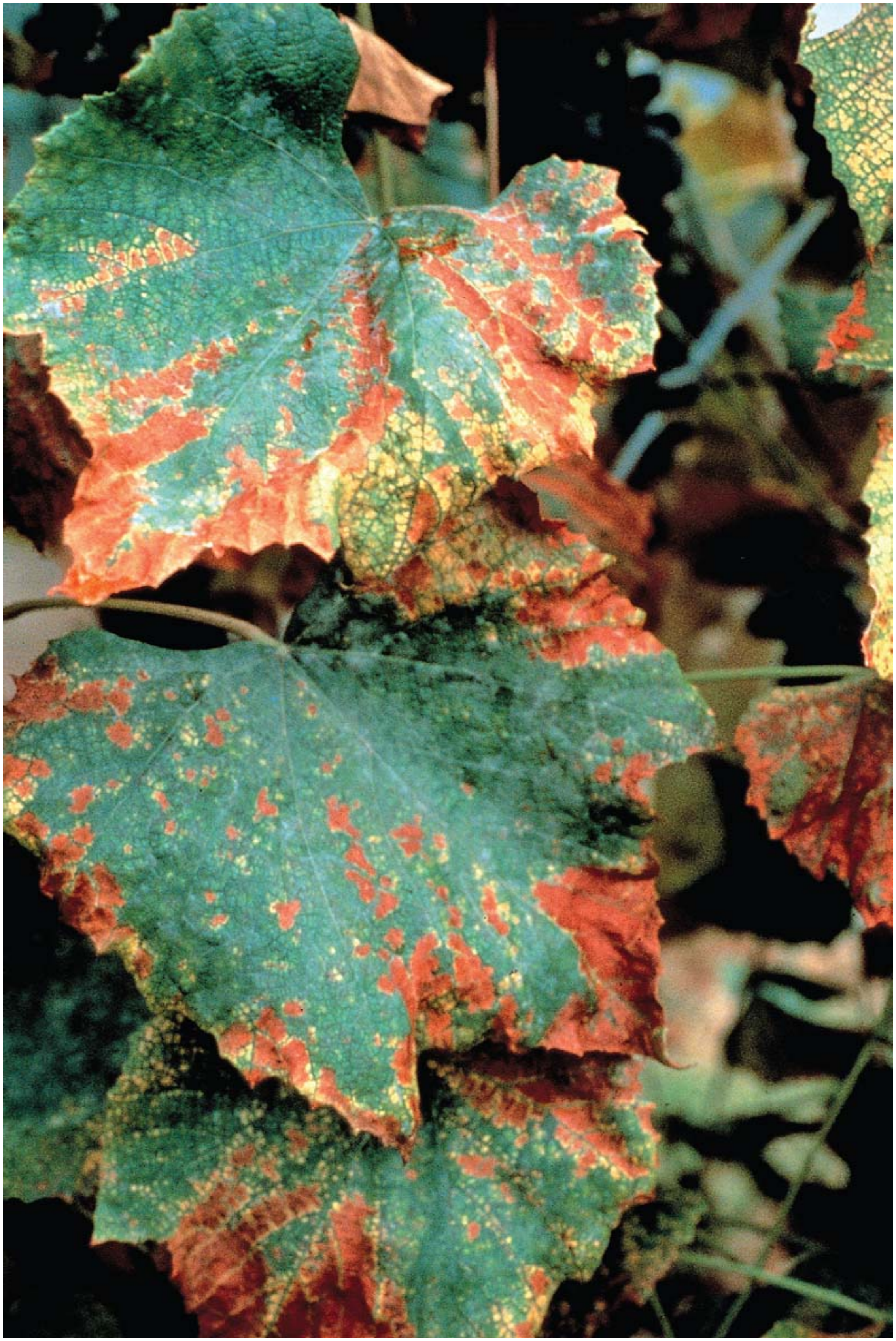


Figure 7.6.
Marginal and interveinal chlorosis and necrosis on grape leaves caused by sulfur dioxide.

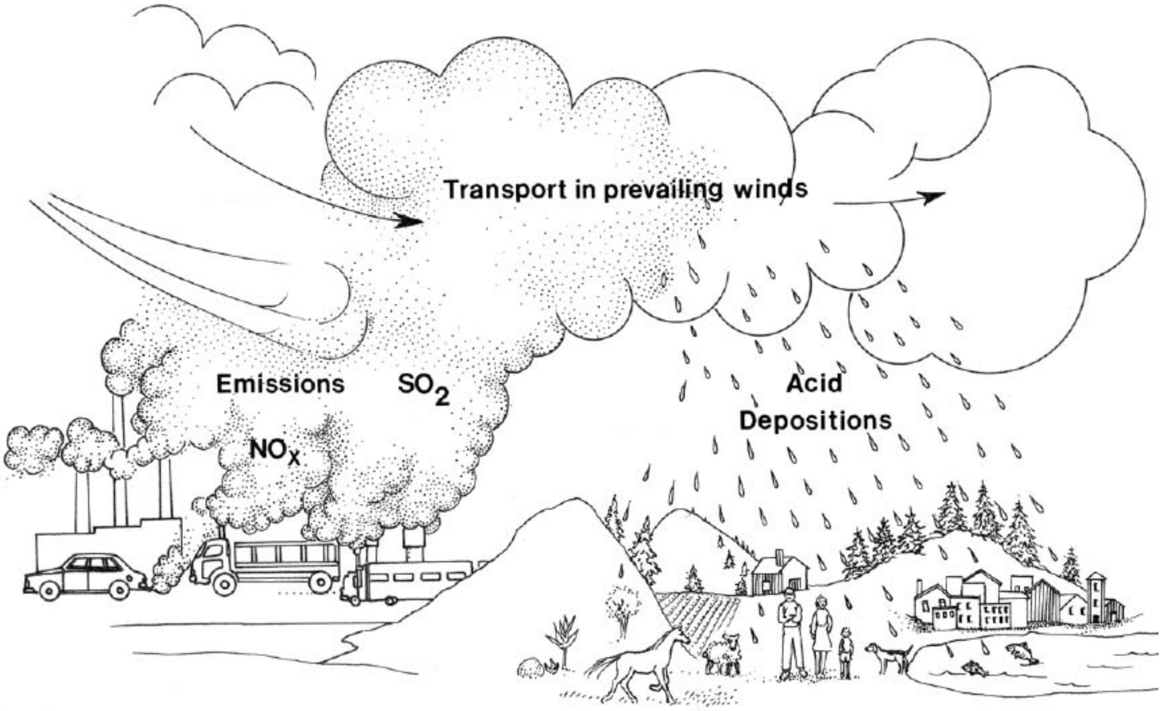

Transport in prevailing winds
Emissions
SO_2
NO_X
Acid
Depositions

Figure 7.7.
Origins of acid precipitation.

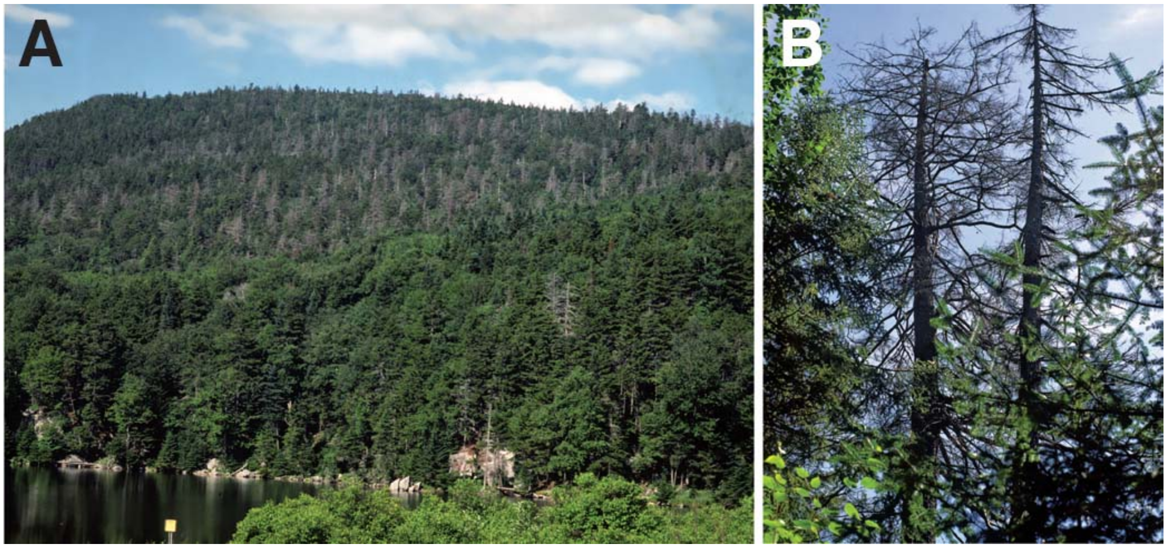


Figure 7.8.
Forest decline of red spruce. **A,** Red spruce mortality, Crawford Notch, White Mountain National Forest, NH. **B,** Dead red spruce, Santanoni Peak, Adirondack Park, NY.

Figure 7.9.
Marginal and interveinal necrosis on grape leaves caused by hydrogen fluoride emitted from an aluminum-processing plant.

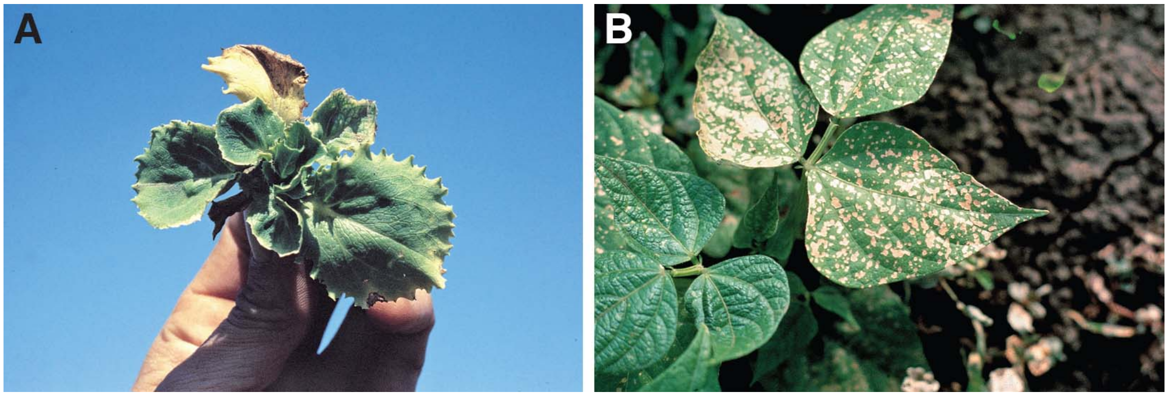


Figure 7.10.
Damage from herbicides due to (**A**) misapplication of glyphosate on lettuce and (**B**) drift of paraquat on beans.

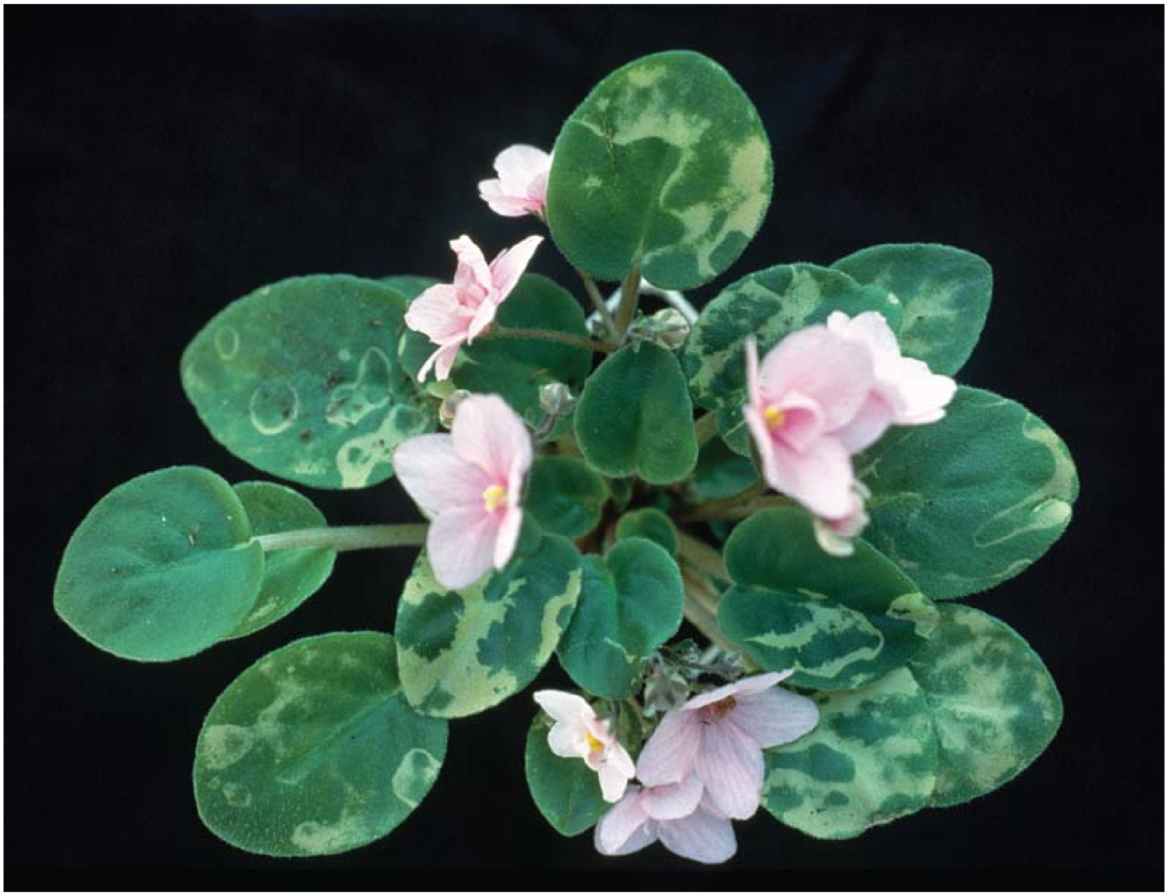


Figure 7.11.
Ring spots on African violet leaves injured by cold water, resembling symptoms of virus infection.

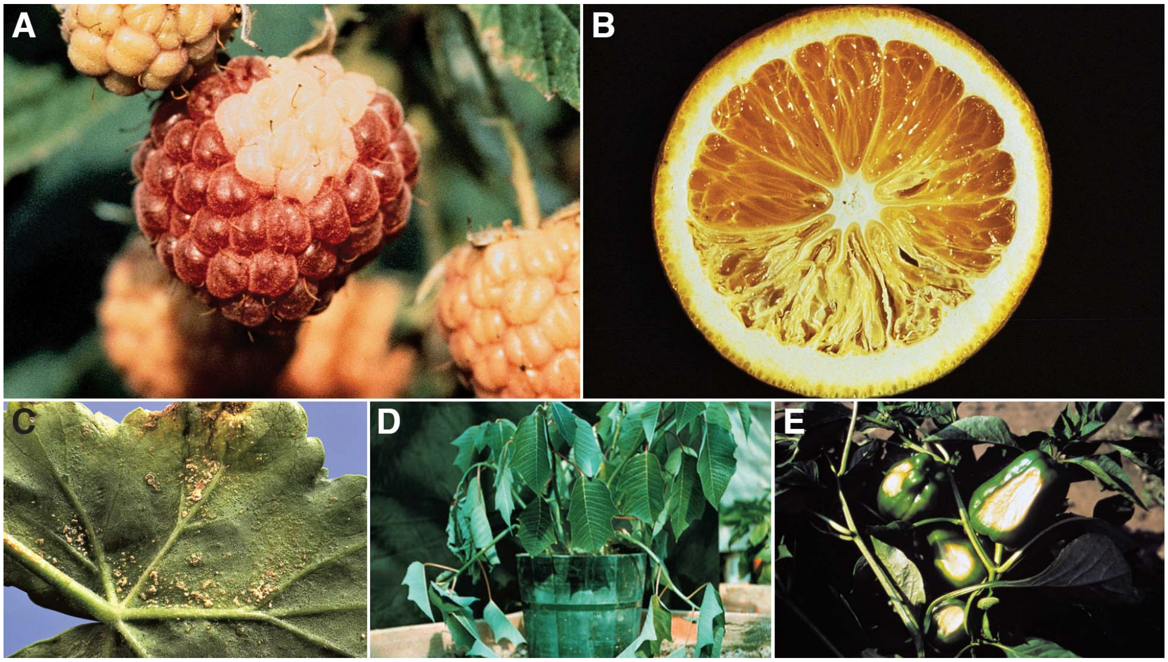


Figure 7.12.

Symptoms caused by environmental factors. **A,** White drupelet disorder of red raspberry caused by high temperatures and ultraviolet radiation. **B,** Freeze injury on orange fruit. **C,** Edema on geranium caused by high relative humidity, high soil temperature, and overwatering. **D,** Wilt on poinsettia caused by lack of water or by root-infecting pathogens. **E,** Sunscald on pepper.

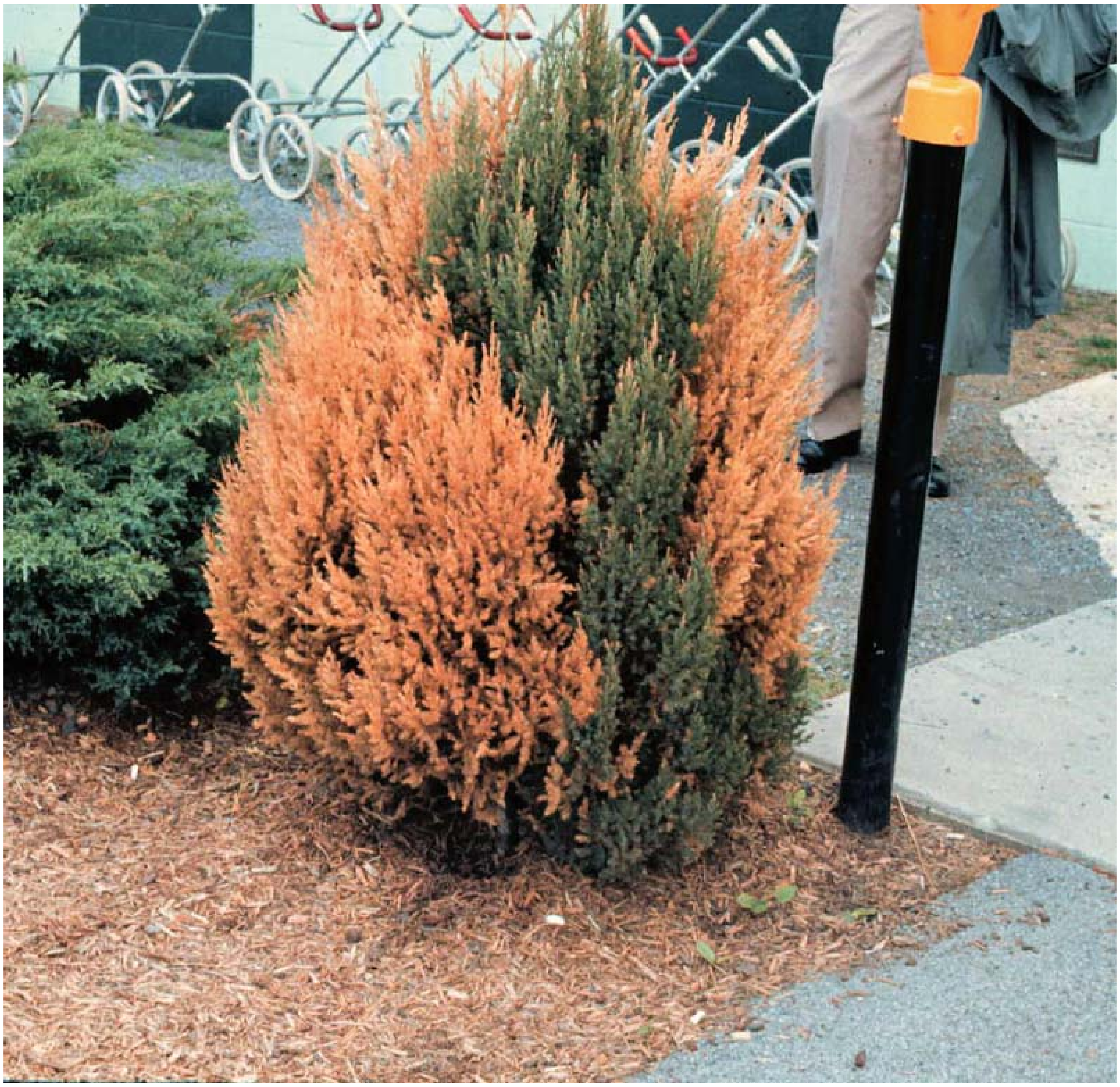


Figure 7.13.
Winterburn on juniper.

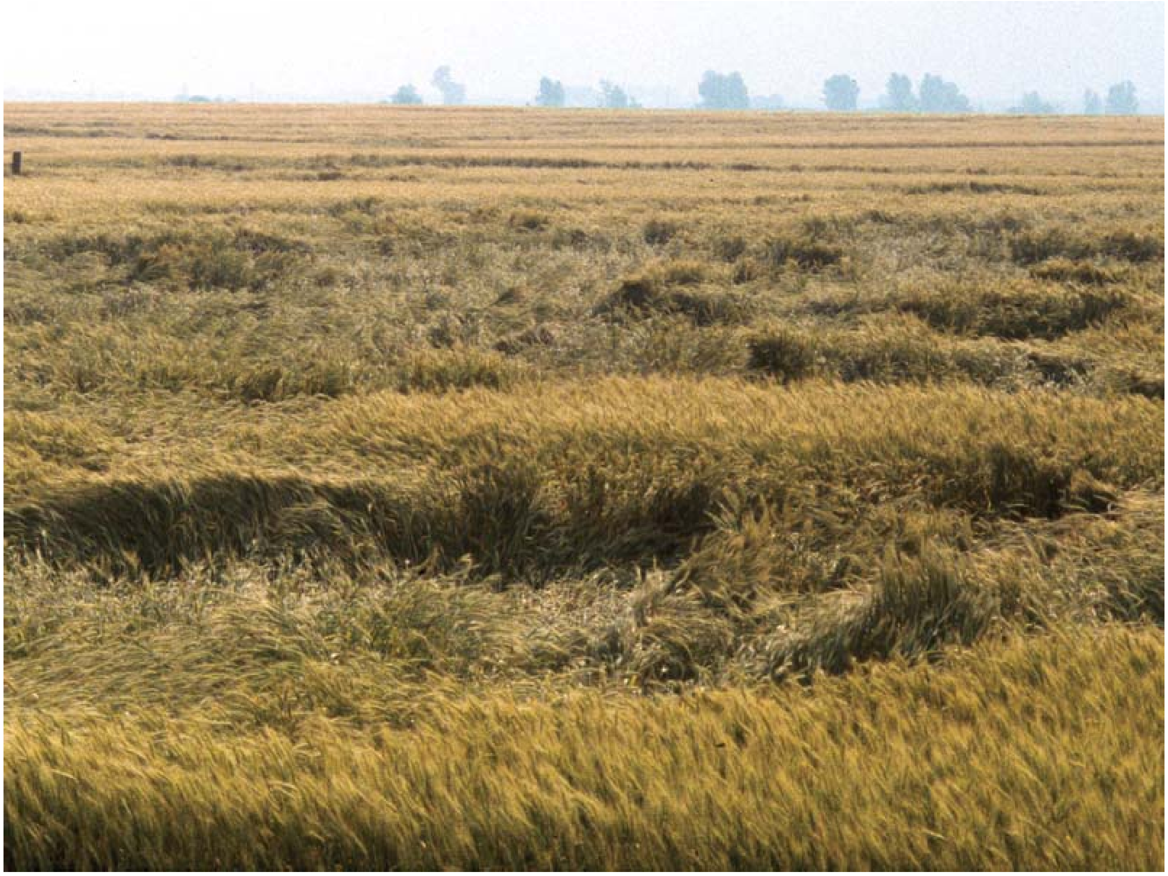


Figure 7.14.
Lodging in winter wheat.

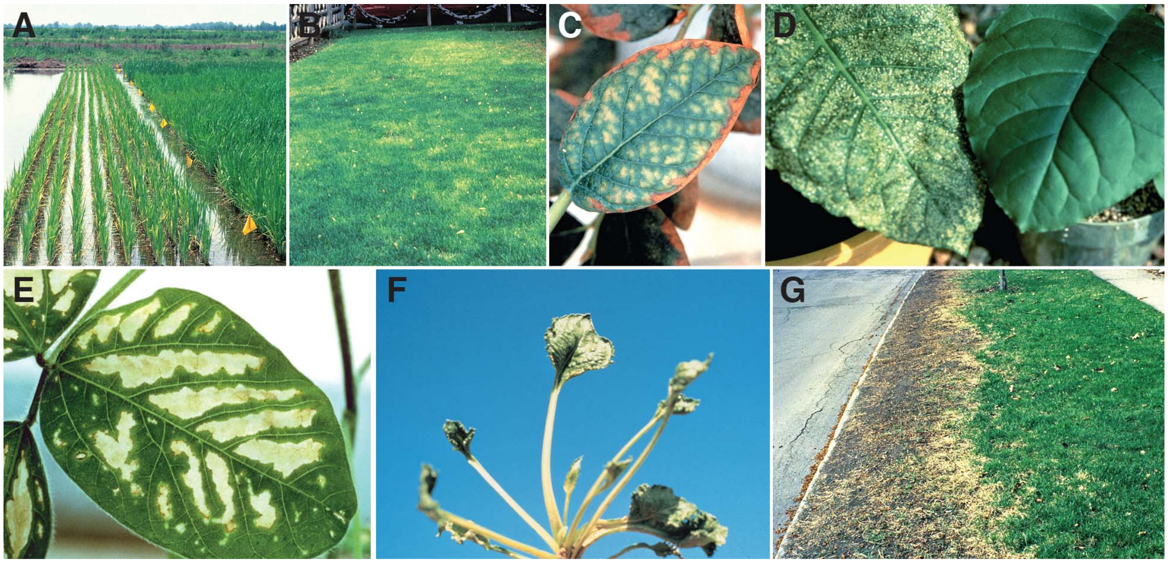


Figure 7.15.
Symptoms caused by chemicals. **A,** Nitrogen deficiency in rice (left). **B,** Iron deficiency in bluegrass. **C,** Boron toxicity in highbush blueberry. **D,** Ozone flecking on tobacco (left). **E,** Acute sulfur dioxide injury on soybean. **F,** Epinasty of sugar beet caused by the herbicide picloram. **G,** Salt toxicity in turfgrass.

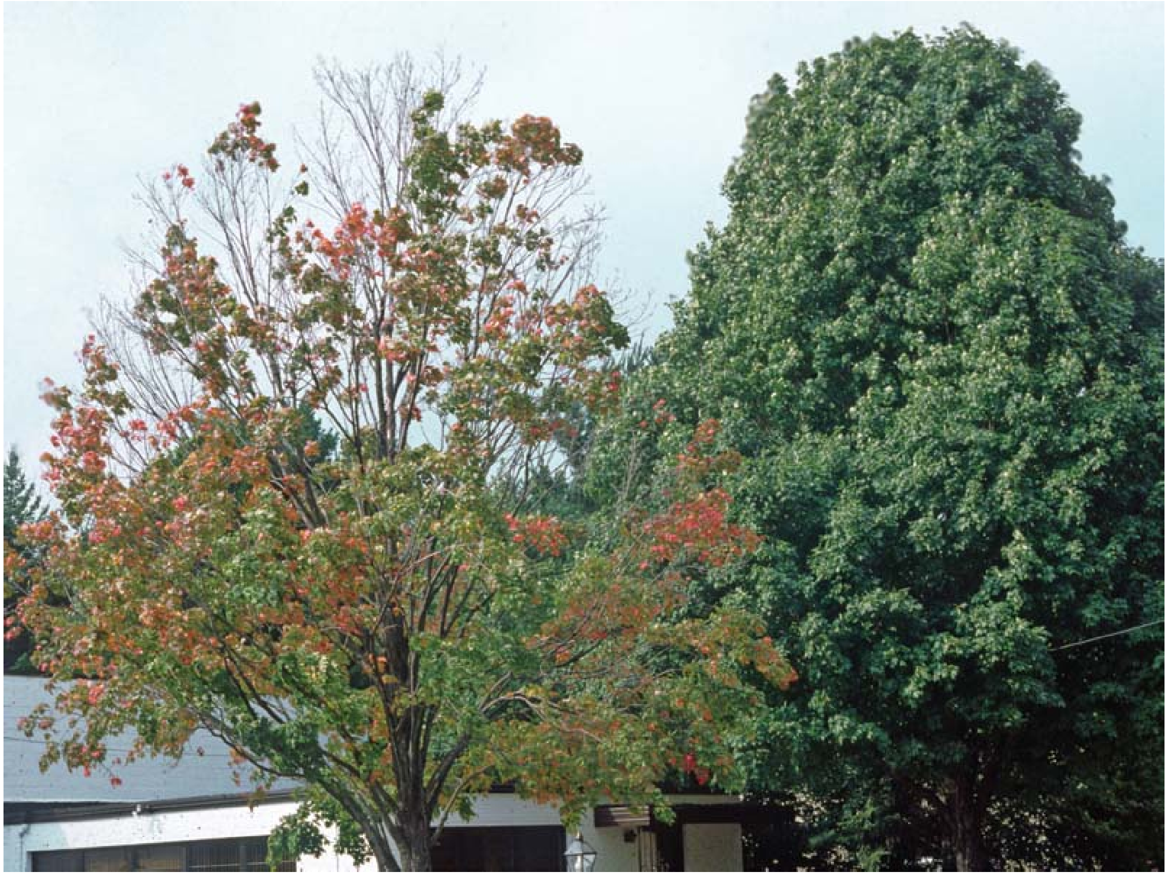


Figure 7.16.
A sugar maple stressed by parking lot paving (left) has developed Armillaria root disease.

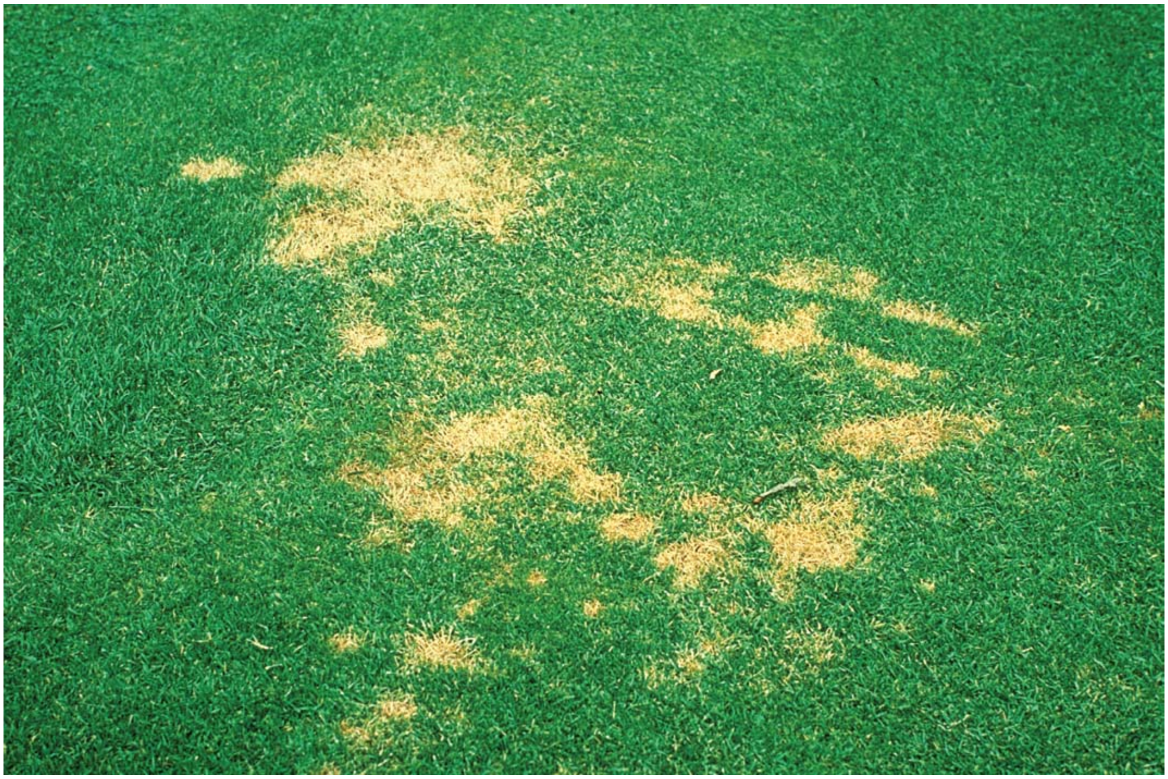


Figure 7.17.
Phytotoxicity from fungicide spilled on turfgrass.

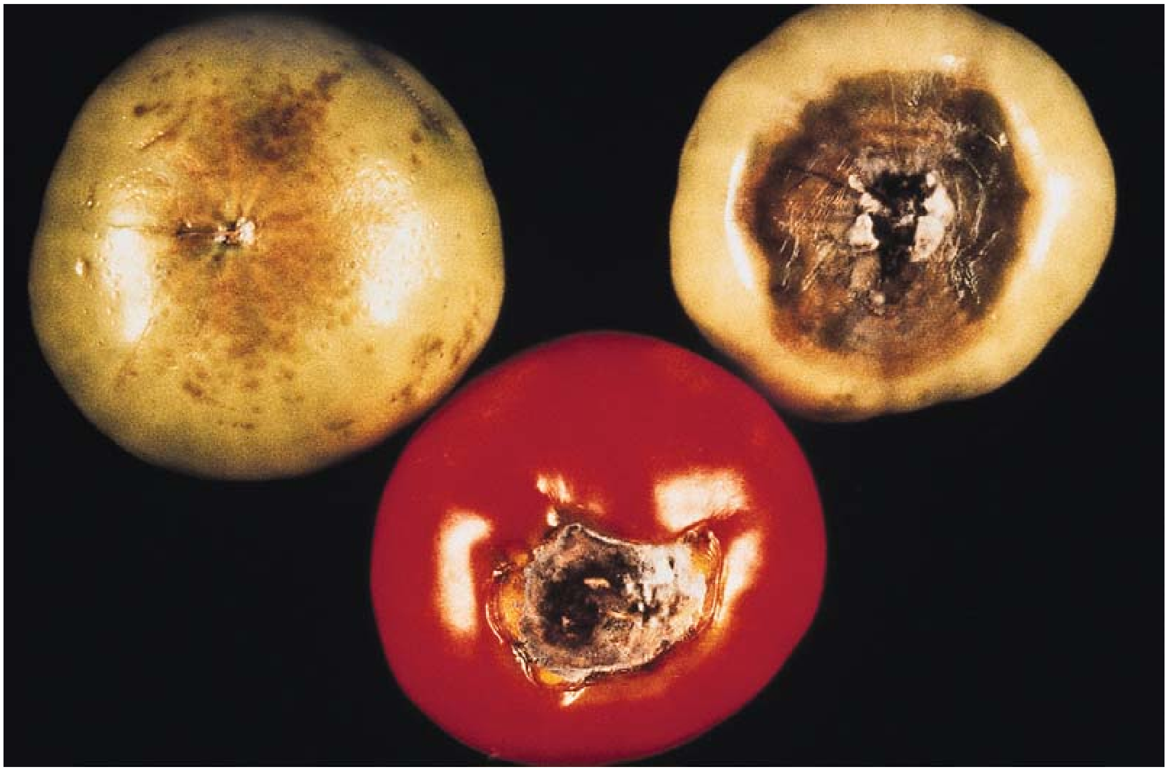


Figure 7.18.
Blossom-end rot of tomato caused by calcium deficiency.

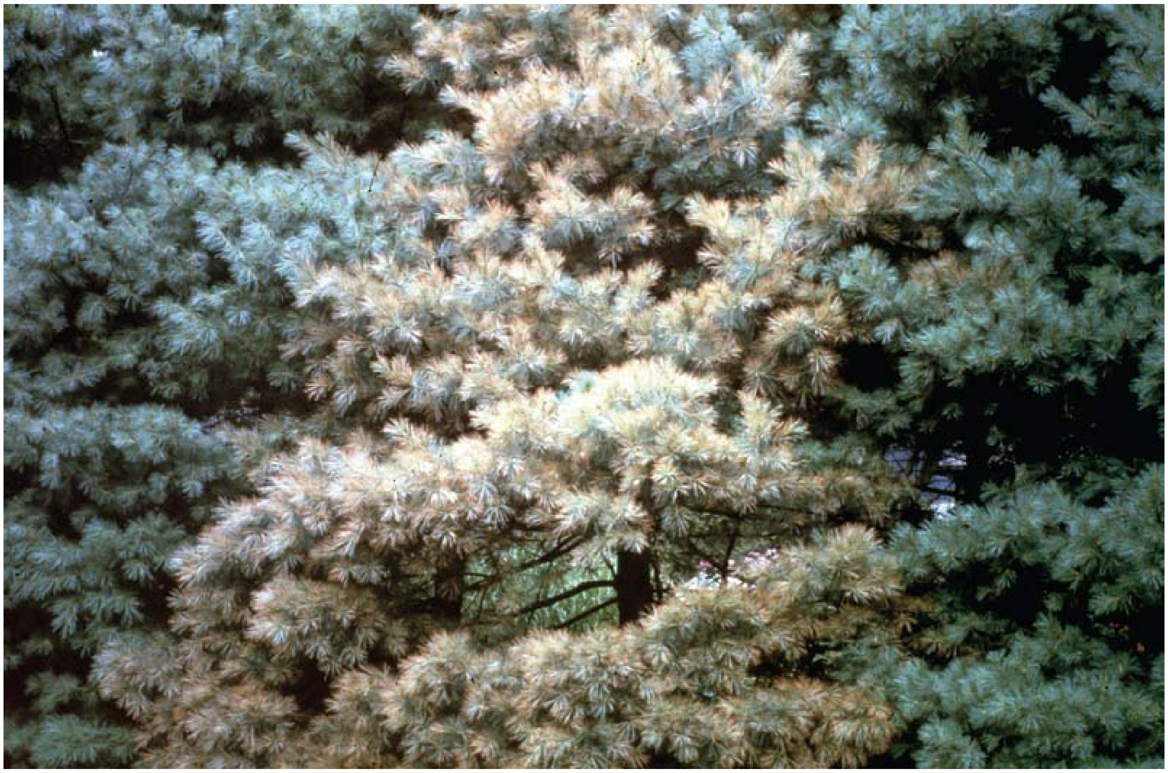


Figure 7.19.
Genetically based variation in sensitivity to ozone in white pine.

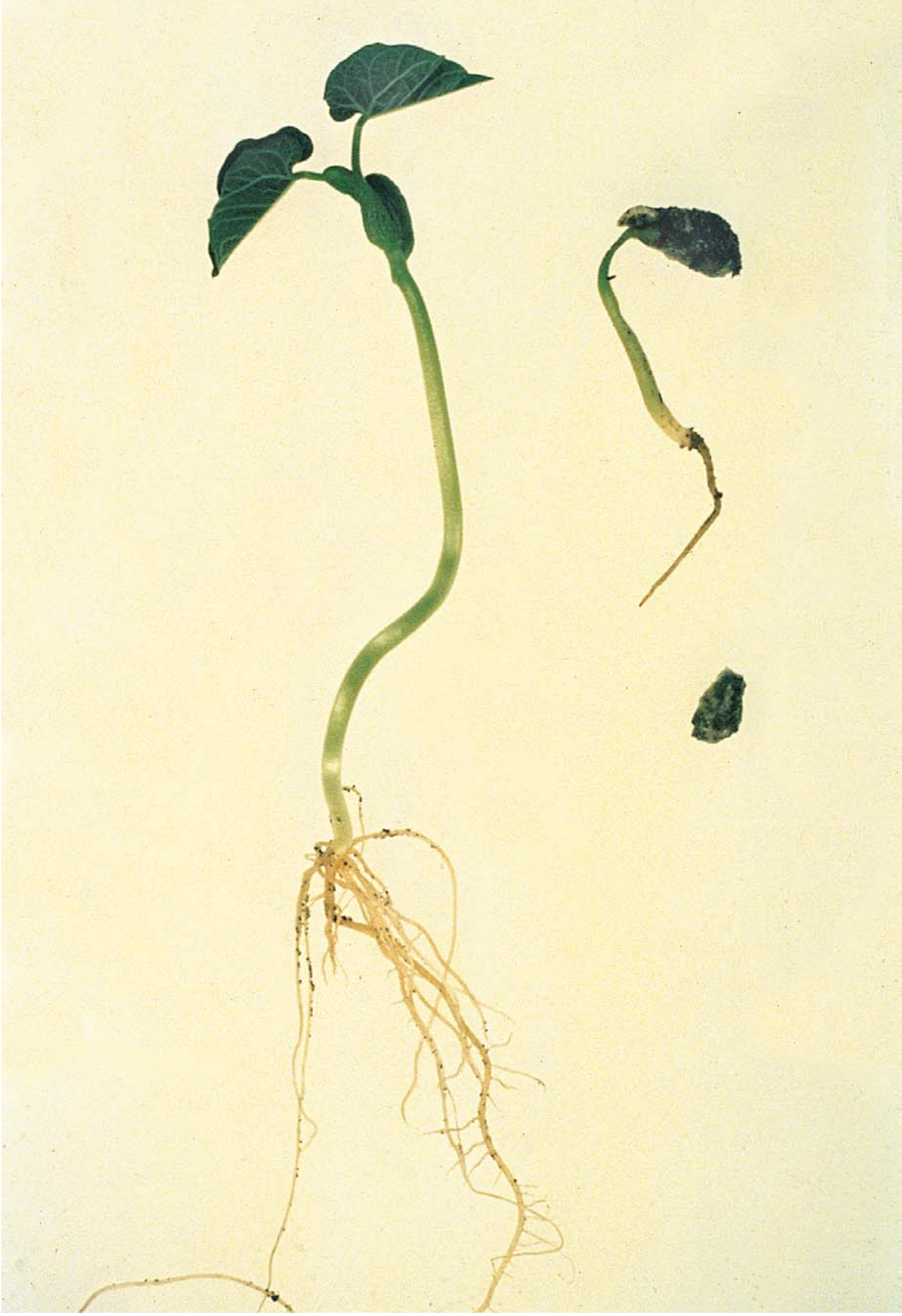


Figure 8.1.
Bean seedling affected by damping-off (right) and a healthy seedling (left). Damping-off includes seed rot and pre- and post-emergence seedling death.

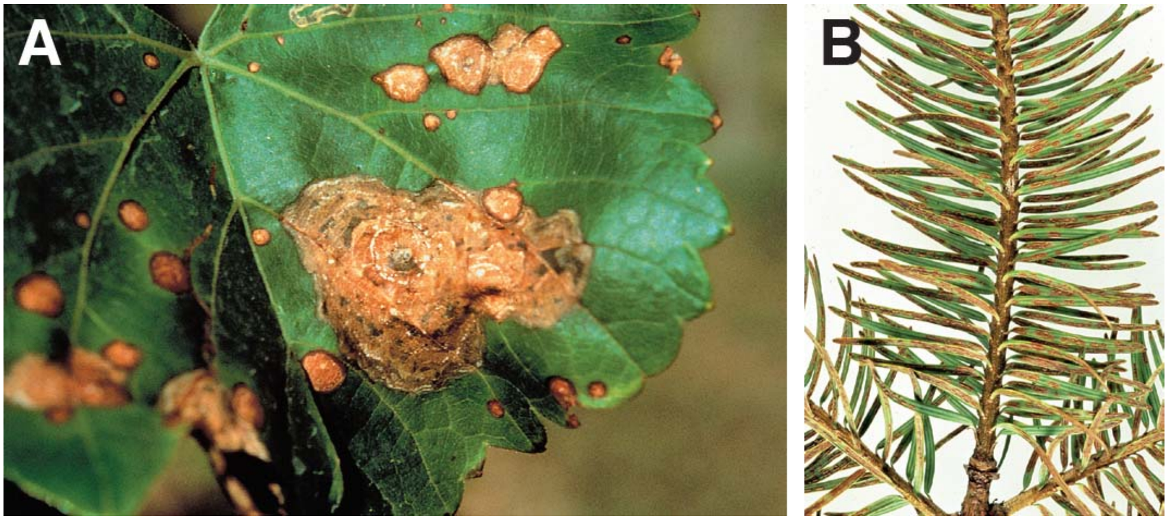


Figure 8.2.
Examples of necrotic foliar symptoms. **A,** Leaf blotch. **B,** Needlecast; infected needles are eventually cast from the tree (i.e., they fall off).

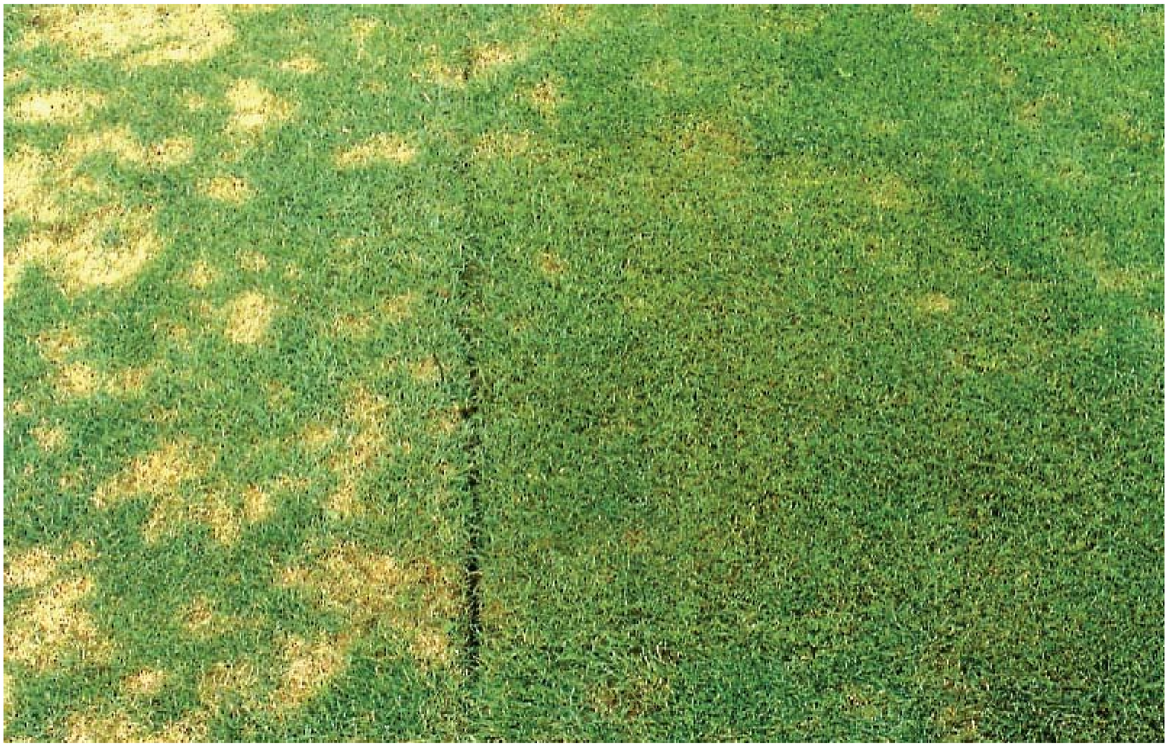


Figure 8.3.
Nitrogen levels can affect susceptibility to disease. Brown patch (right) occurs in turfgrass in places where high levels of nitrogen fertilizer were applied. Dollar spot (left) occurs where nitrogen is deficient.

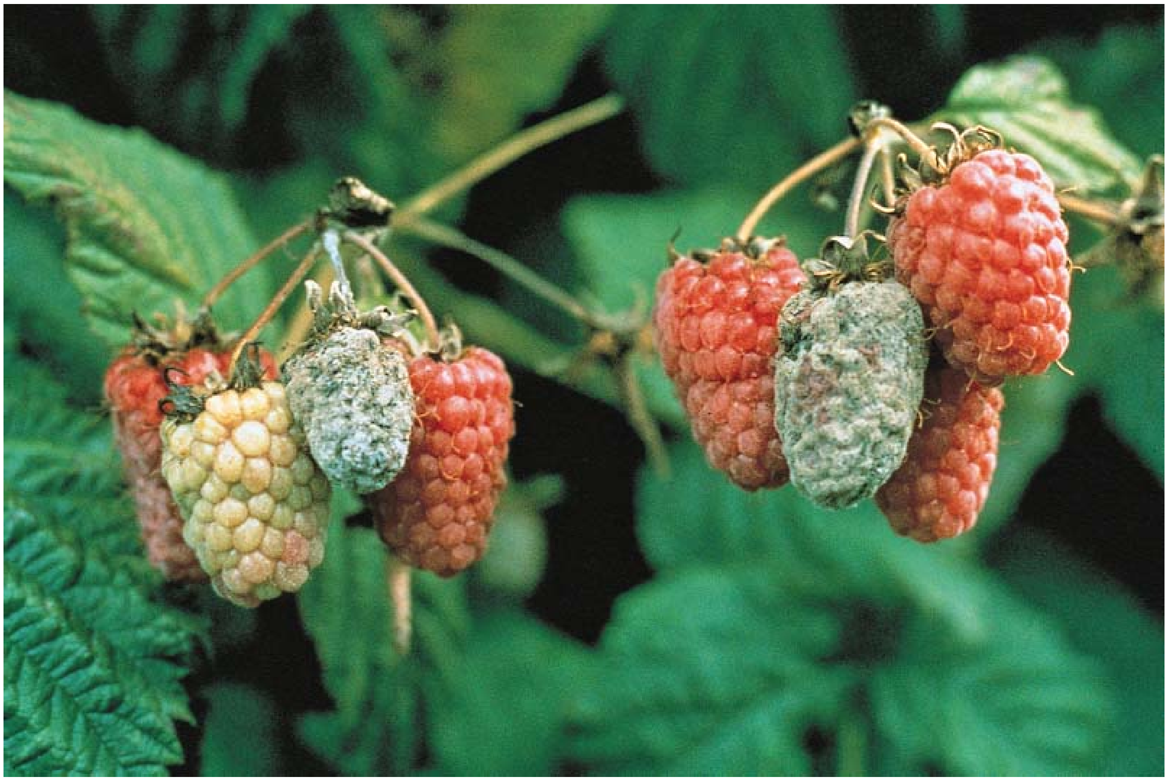
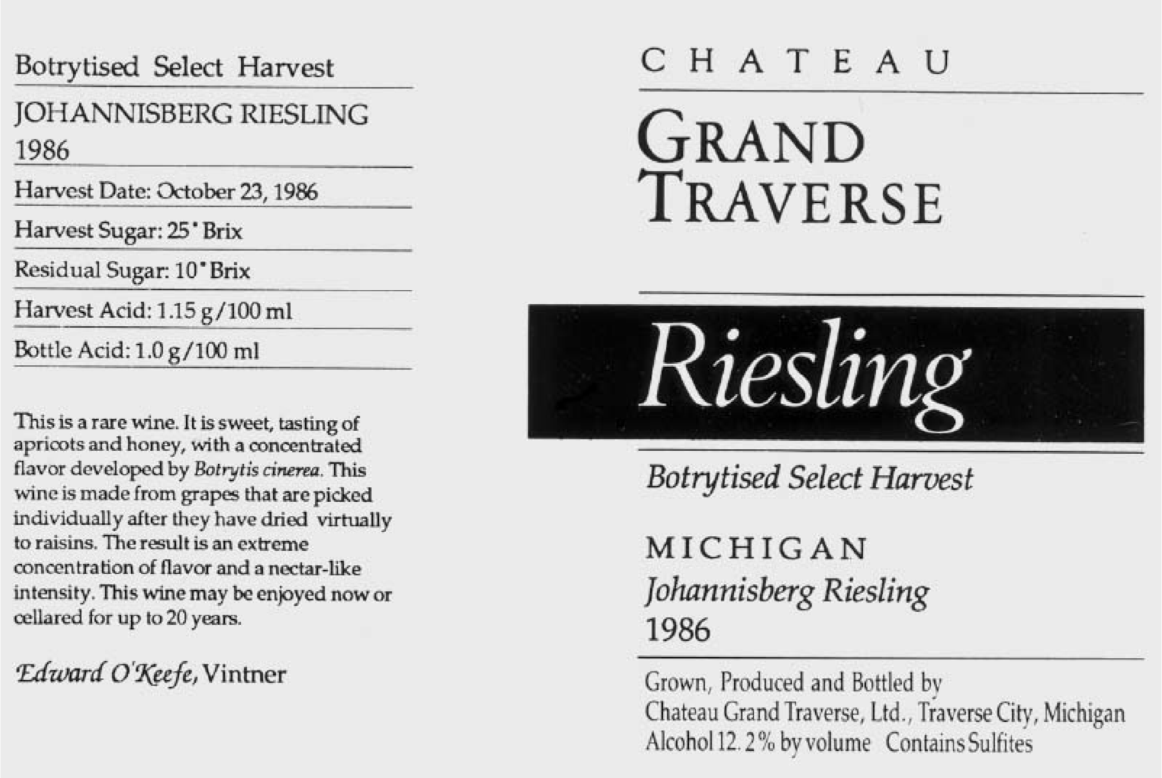


Figure 8.4.

Gray mold, caused by *Botrytis cinerea*, on red raspberry fruit. Note the dusty covering of gray spores on infected fruit.



Botrytised Select Harvest

JOHANNISBERG RIESLING
1986

Harvest Date: October 23, 1986

Harvest Sugar: 25° Brix

Residual Sugar: 10° Brix

Harvest Acid: 1.15 g/100 ml

Bottle Acid: 1.0 g/100 ml

This is a rare wine. It is sweet, tasting of apricots and honey, with a concentrated flavor developed by *Botrytis cinerea.* This wine is made from grapes that are picked individually after they have dried virtually to raisins. The result is an extreme concentration of flavor and a nectar-like intensity. This wine may be enjoyed now or cellared for up to 20 years.

Edward O'Keefe, Vintner

CHATEAU
GRAND
TRAVERSE

Riesling

Botrytised Select Harvest

MICHIGAN
Johannisberg Riesling
1986

Grown, Produced and Bottled by
Chateau Grand Traverse, Ltd., Traverse City, Michigan
Alcohol 12.2% by volume Contains Sulfites

Figure 8.5.
Label of a wine produced from grapes that were “botrytised,” or infected by *Botrytis cinerea*, the fungus causing “the noble rot.”

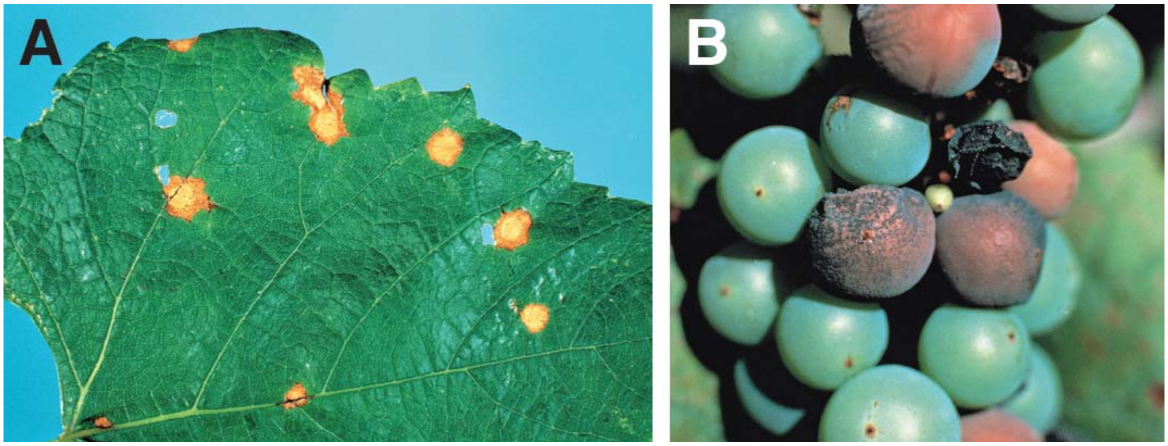


Figure 8.6.
A, Black rot lesions on a grape leaf. **B,** Black rot of a cluster of grapes at various stages of disease.

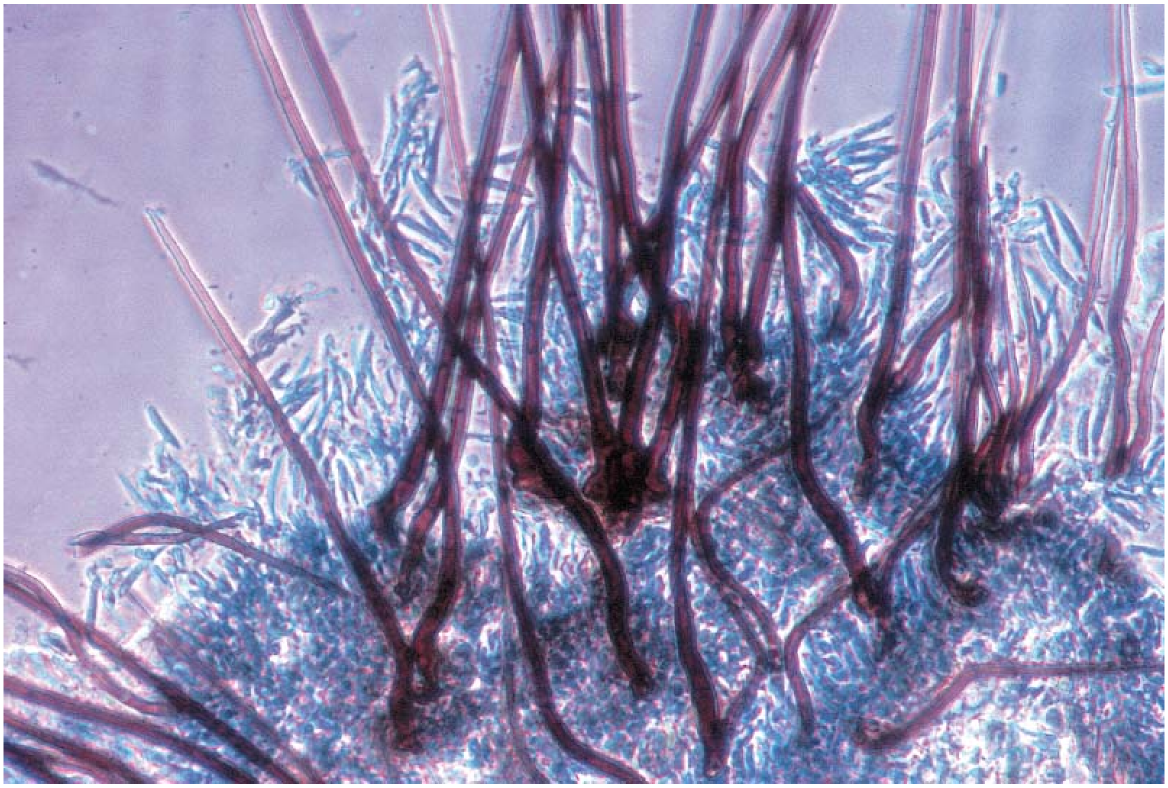


Figure 8.7.
Acervulus of an anthracnose fungus observed with a compound microscope. Note the black setae (hairlike projections) and the light-colored conidia. (x400)

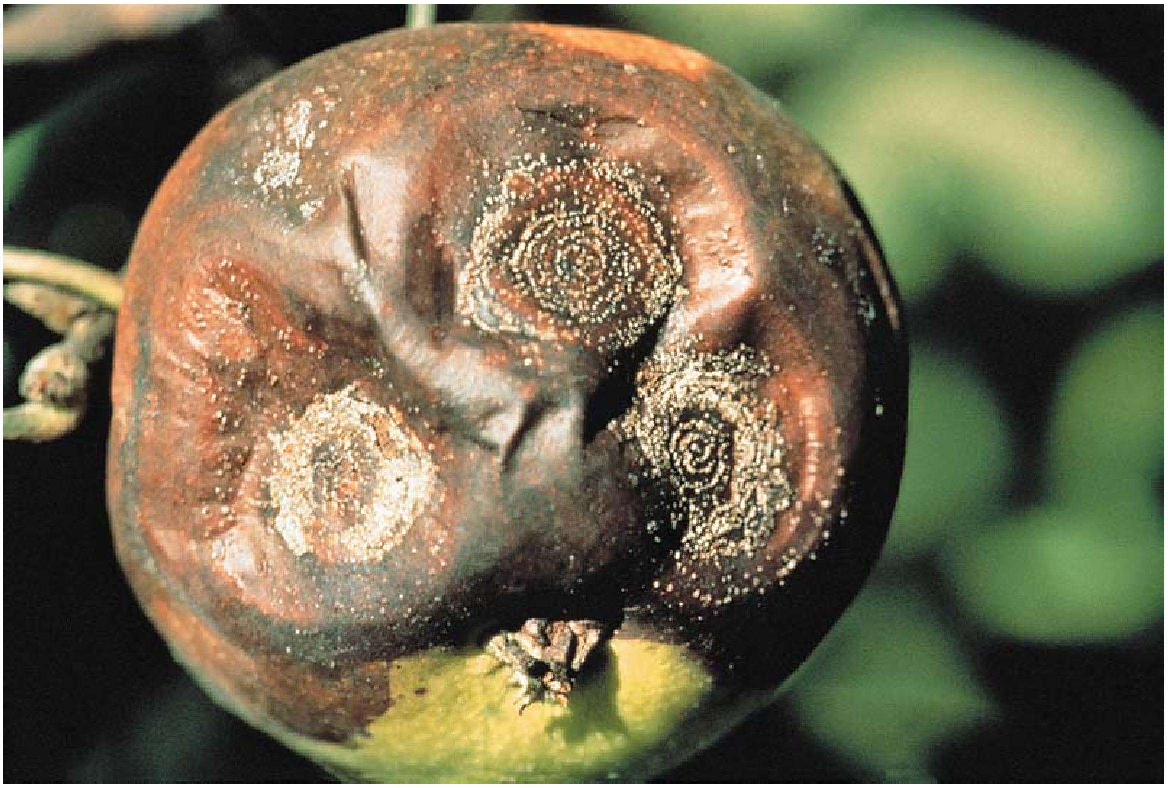


Figure 8.8.
Bitter rot, an anthracnose disease of apple, caused by *Colletotrichum gloeosporioides* and *C. acutatum.* Note the concentric rings of acervuli.

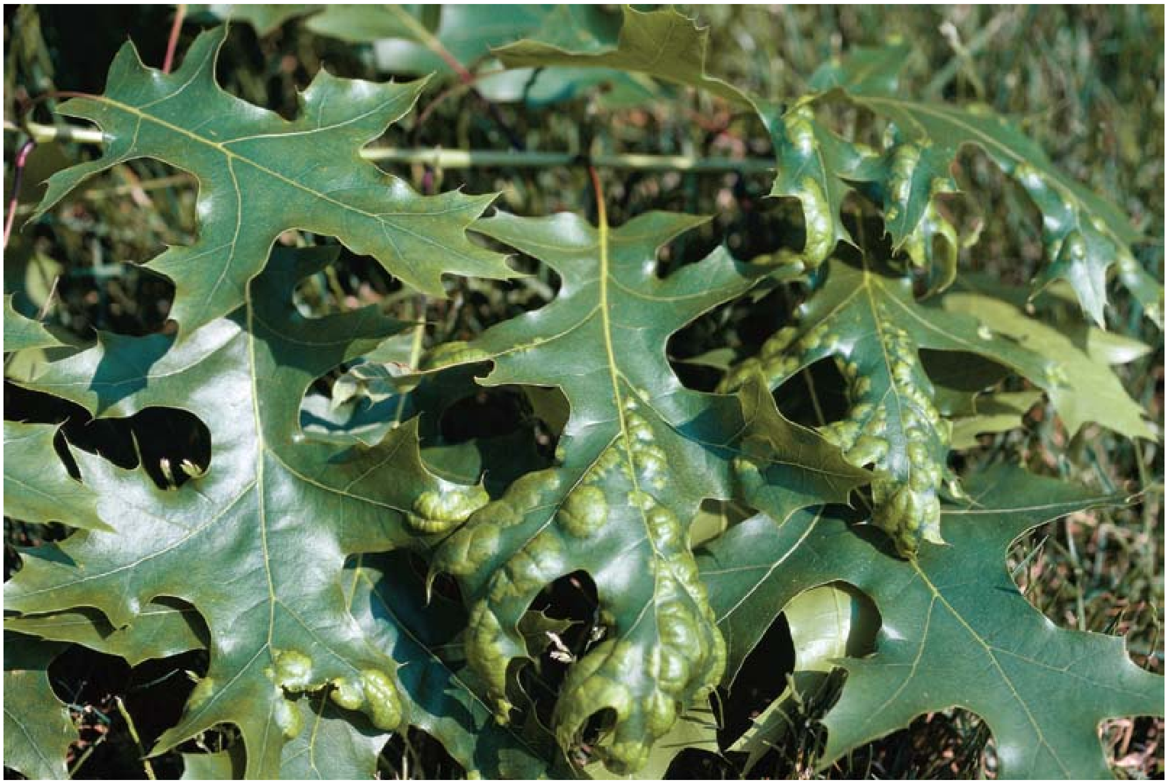


Figure 8.9.
Oak leaf blister, caused by *Taphrina caerulescens*.

Figure 8.10.
Red raspberry with foliar ring spots and line patterns caused by infection by *Tomato ringspot virus.*

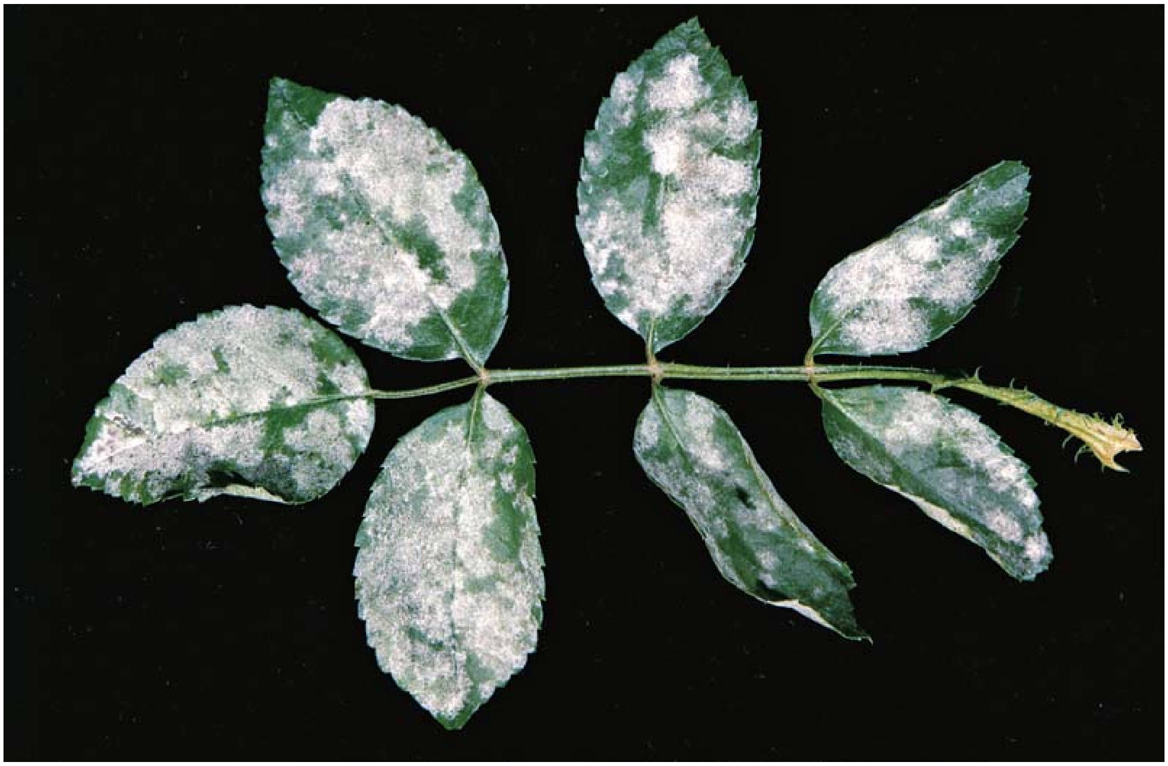


Figure 8.11.
Powdery mildews are named for the powdery white growth of mycelium and conidia on plant surfaces.

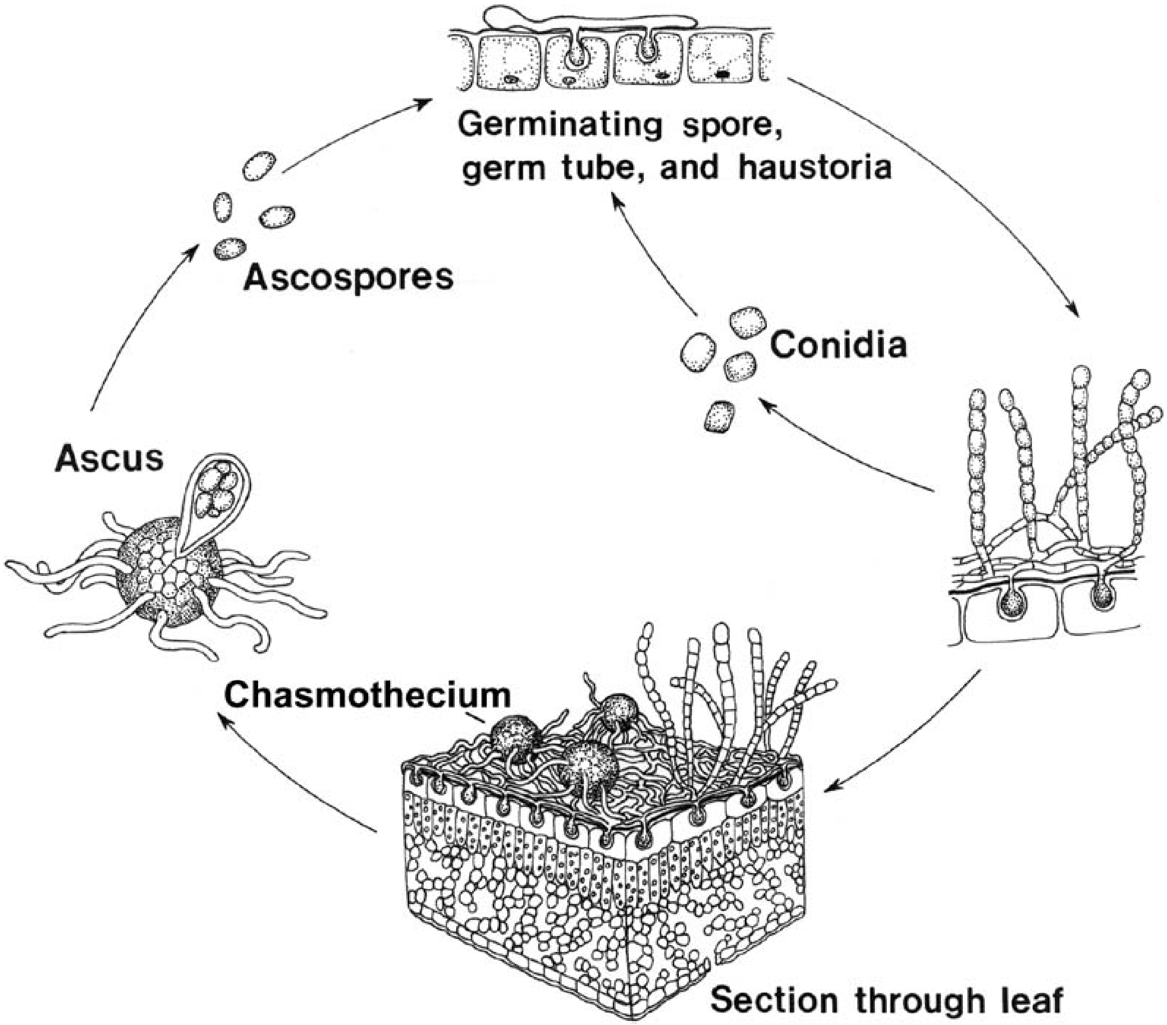

Germinating spore,
germ tube, and haustoria
Ascospores
Conidia
Ascus
Chasmothecium
Section through leaf

Figure 8.12.
Life cycle of a powdery mildew fungus.

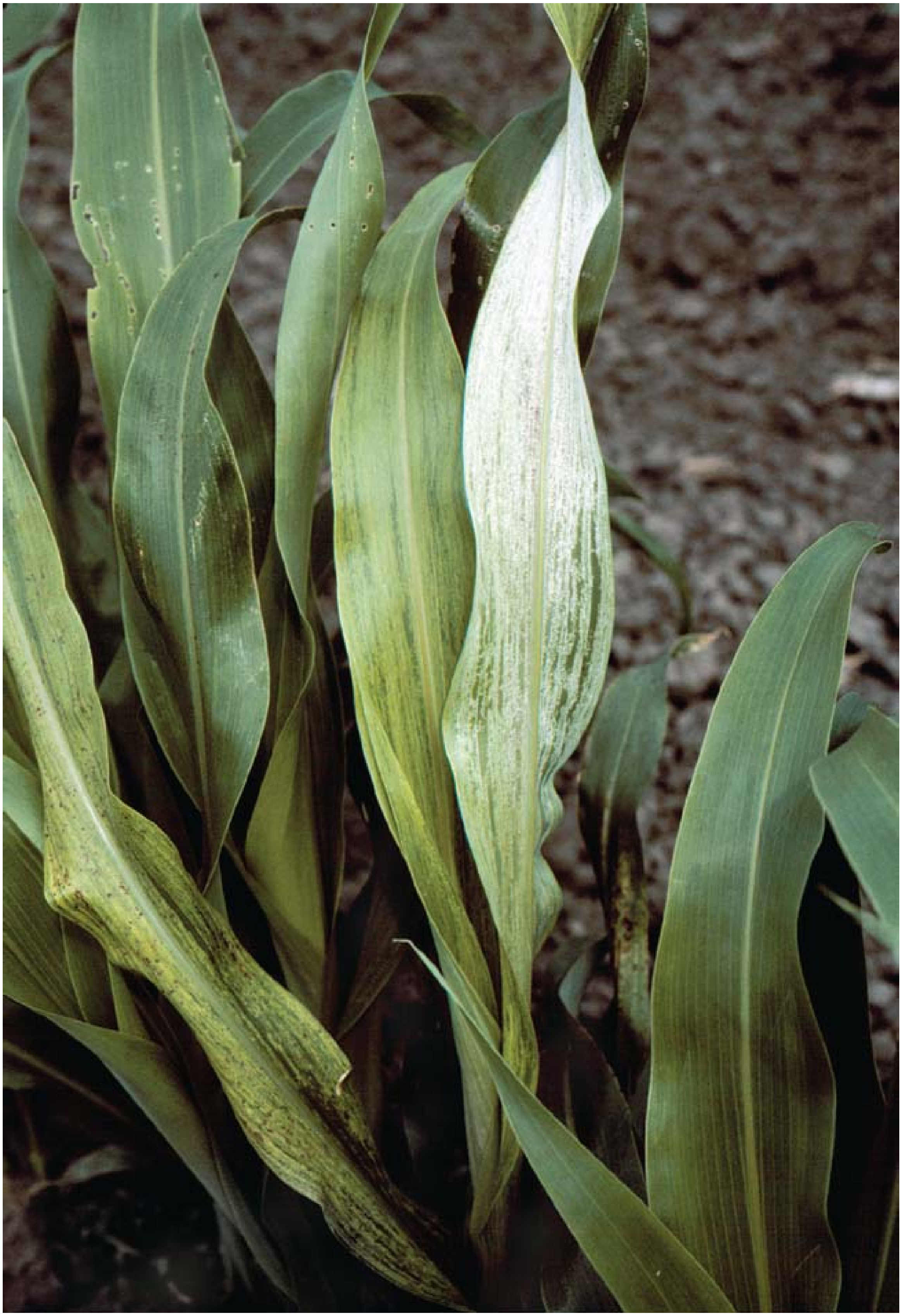


Figure 8.13.
Sporulation of a downy mildew pathogen, *Peronosclerospora sorghi*, on sorghum.

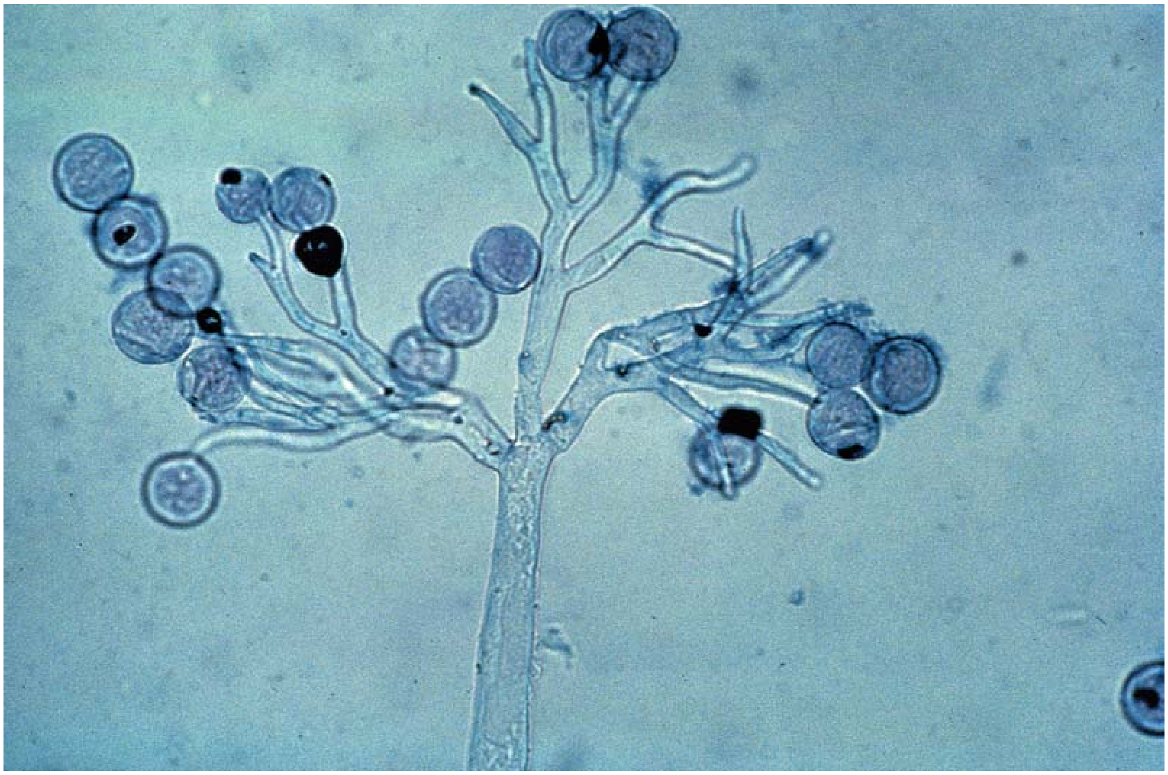


Figure 8.14.
Sporangiophore and sporangia of a downy mildew pathogen.

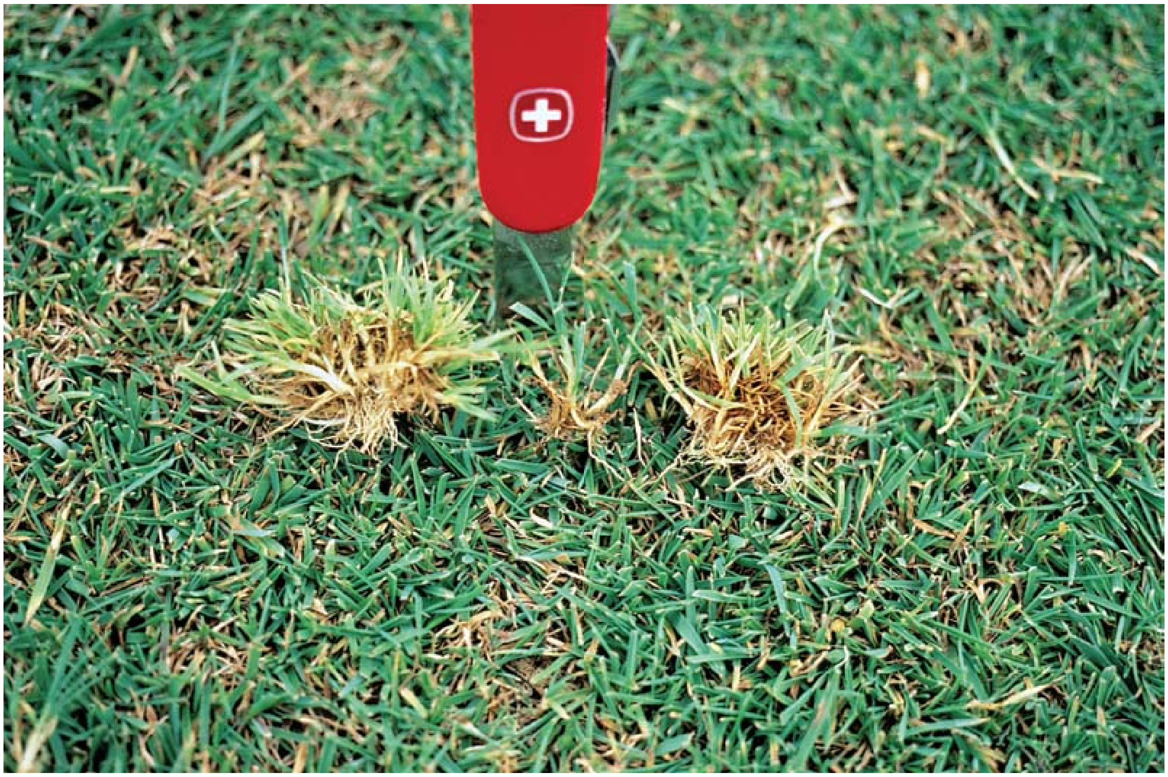


Figure 8.15.
Yellow tuft, a systemic downy mildew disease of turfgrass, caused by *Sclerophthora macrospora*.

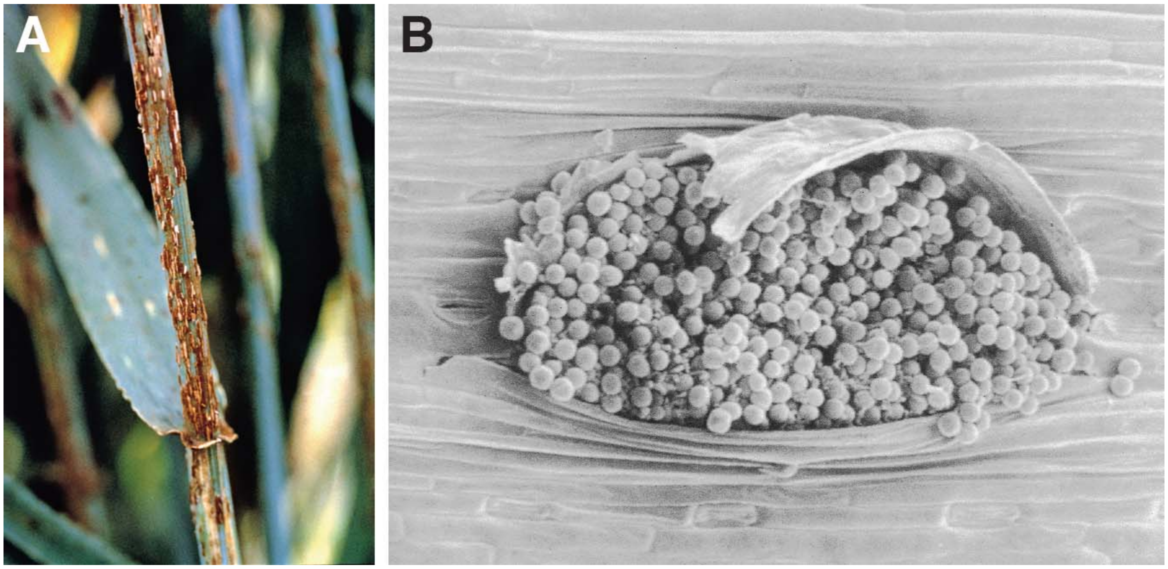


Figure 8.16.
Uredinia (**A**) and orange, powdery urediniospores (**B**) of *Puccinia graminis*, the fungus causing stem rust of wheat.

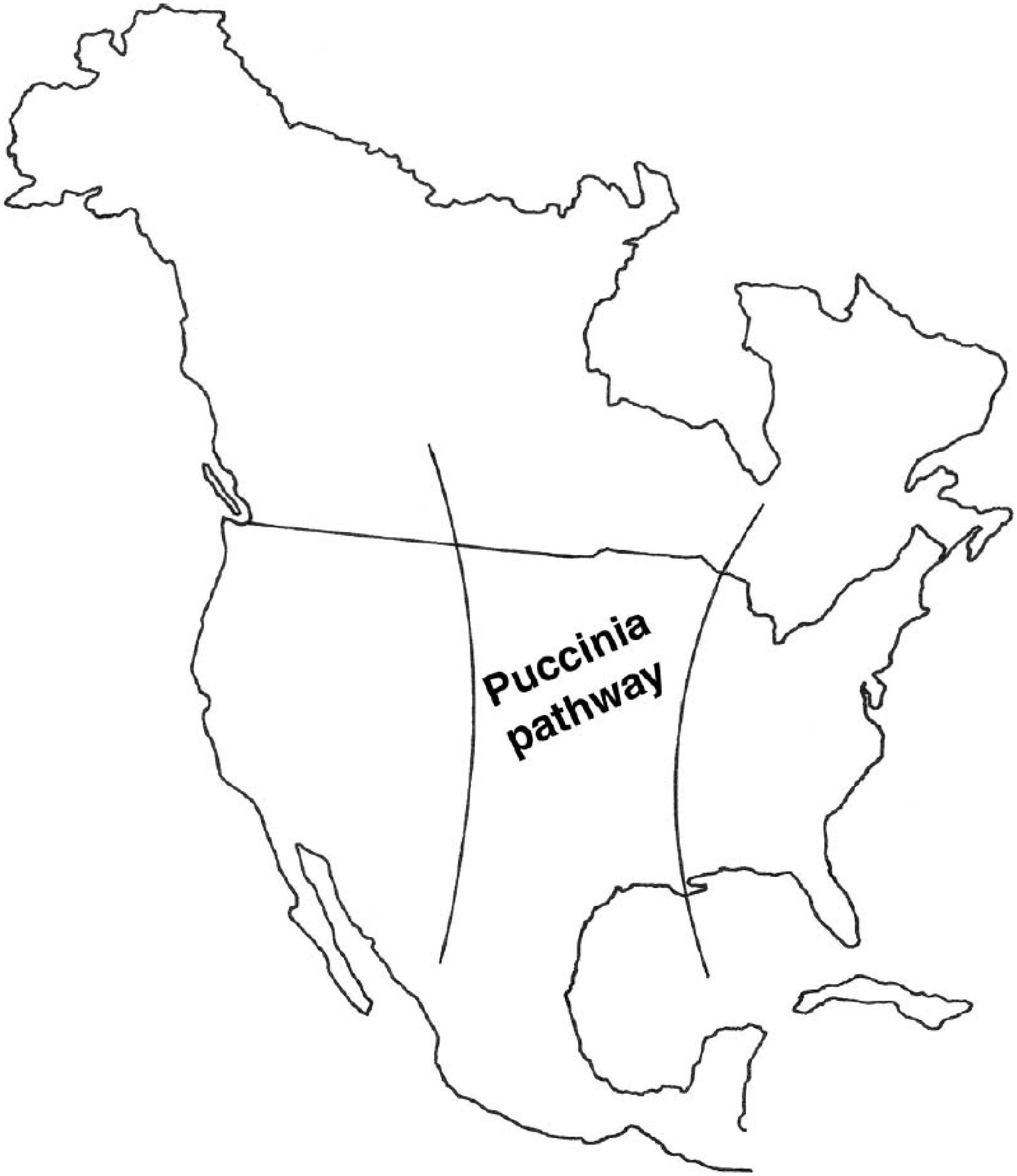


Figure 8.17.
The Puccinia pathway. Each year urediniospores of rust fungi are carried by winds from Mexico and the southern United States across North America to Canada.

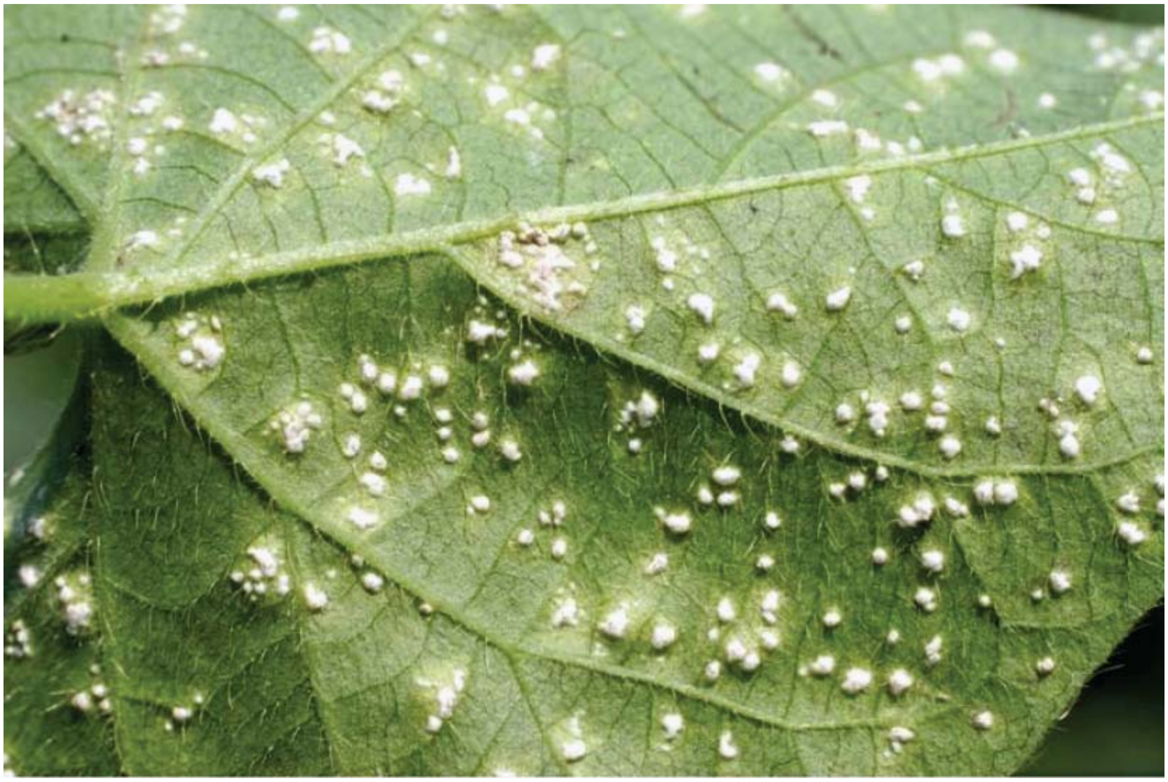


Figure 8.18.
White rust, caused by *Albugo* sp., on a morning-glory leaf (*Ipomoea* sp.).

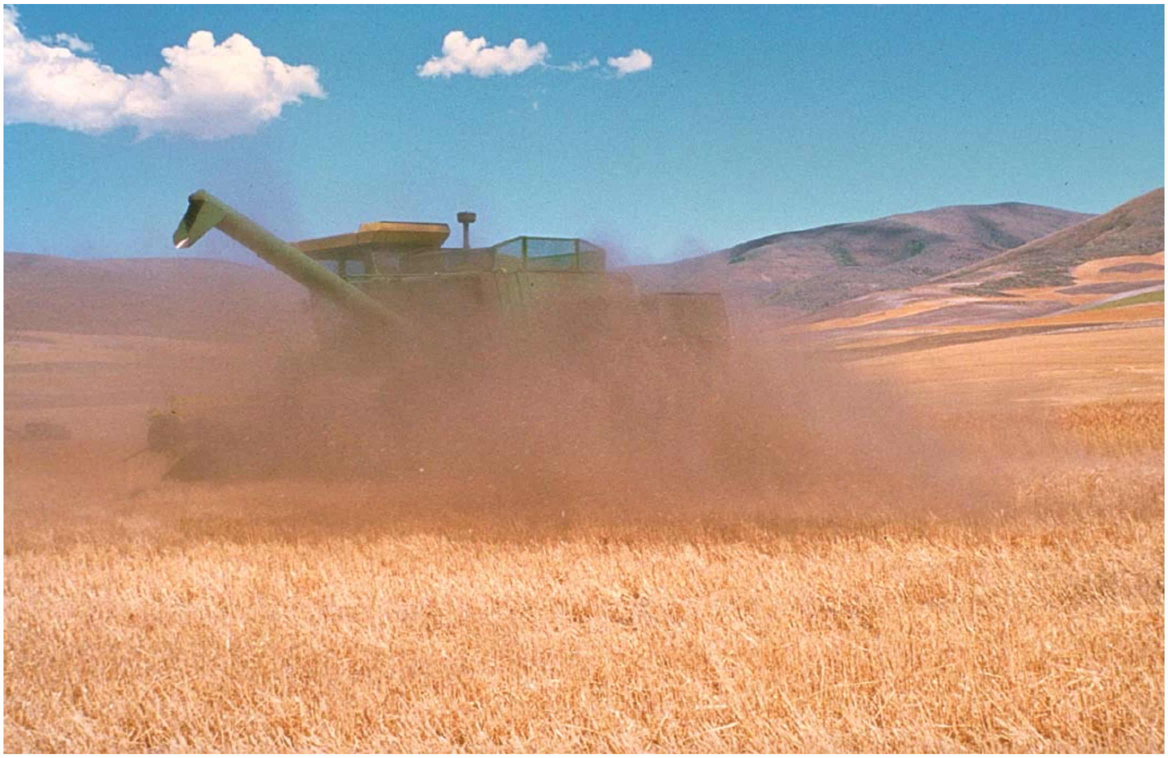


Figure 8.19.
Cloud of smut teliospores during wheat harvest.

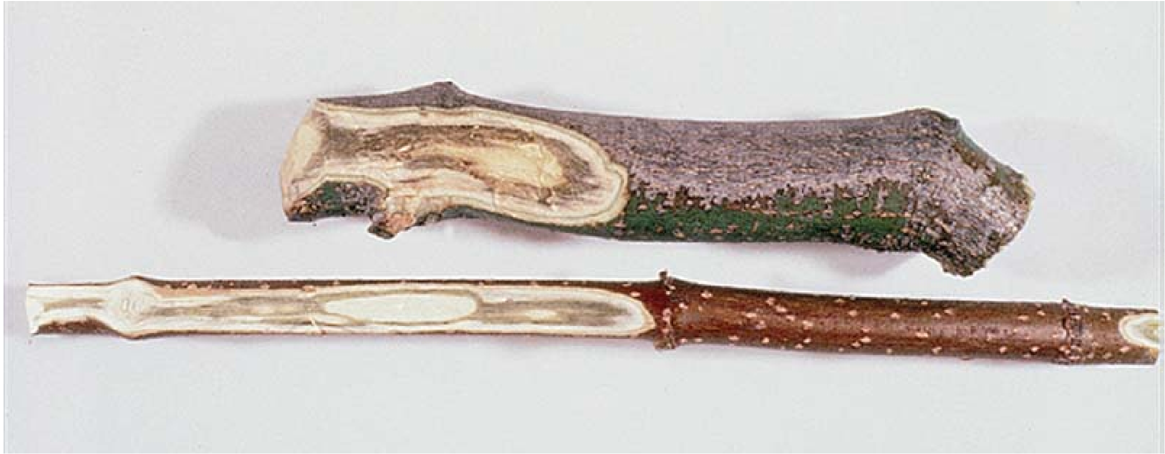


Figure 8.20.
Streaking of vascular tissue due to infection by *Verticillium* sp.

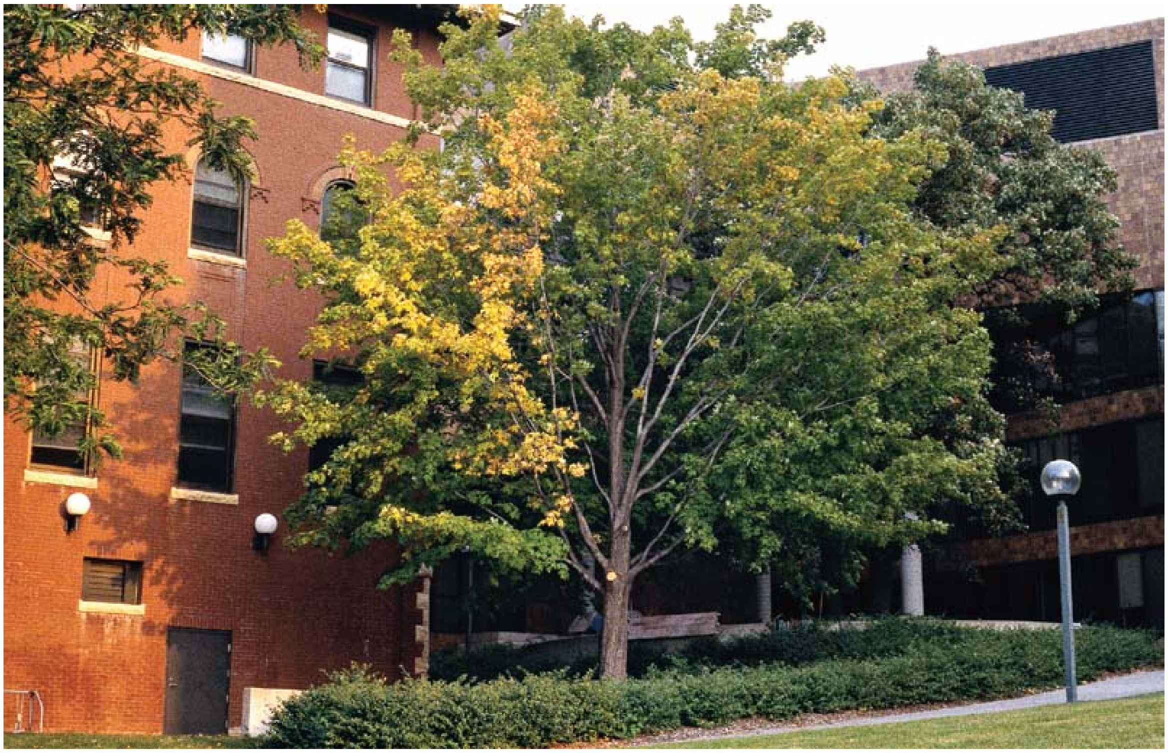


Figure 8.21.
Sugar maple with branch dieback due to Verticillium wilt. Note the one-sided symptoms.

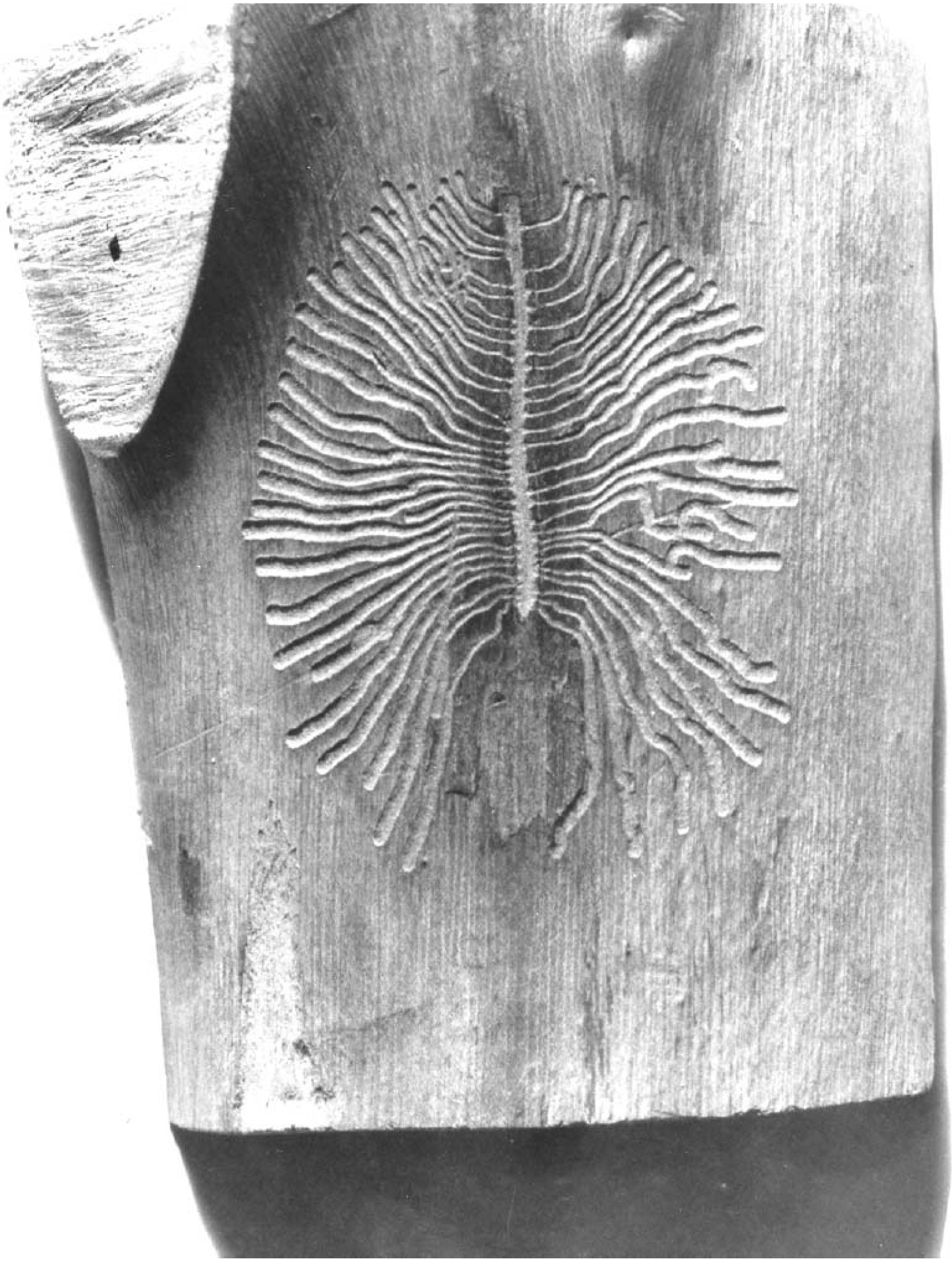


Figure 8.22.
Egg gallery of the European elm bark beetle. Eggs are laid in the maternal tunnel, under the bark and parallel to the grain of the wood.

Synnemata of *Ophiostoma ulmi* are produced in the larval tunnels, which are perpendicular to the maternal tunnel and produced by the feeding larvae.

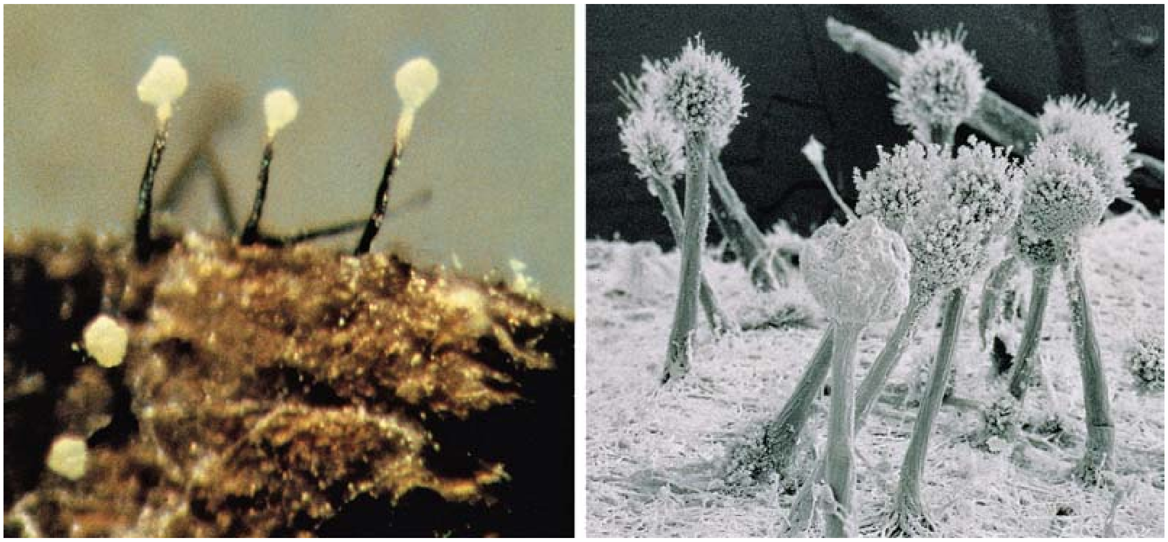


Figure 8.23.
Light microscope (left) and scanning electron microscope (right) views of synnemata of *Ophiostoma ulmi*, one of the fungi causing Dutch elm disease. The conidia stick to adult elm bark beetles as they emerge from egg galleries (see Figure 8.22).

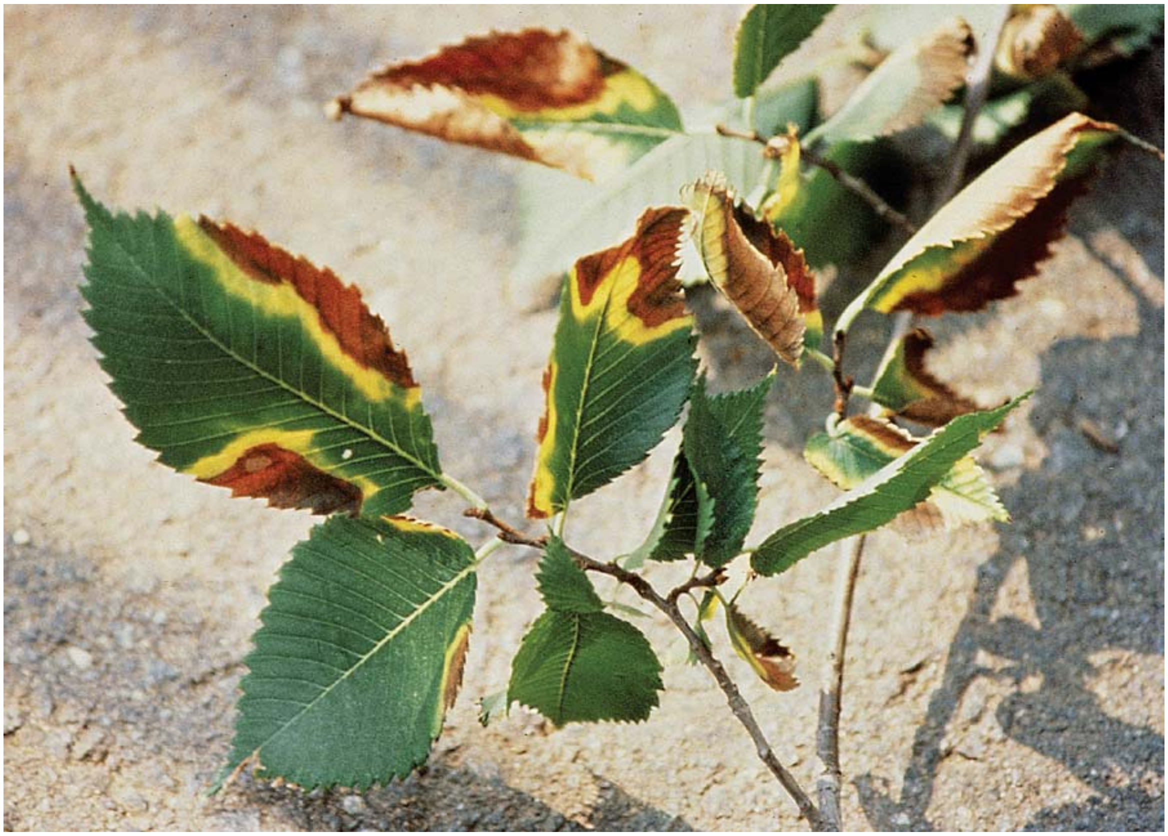


Figure 8.24.
Elm leaf scorch, caused by *Xylella fastidiosa*. Note the undulating marginal necrosis with a chlorotic halo.

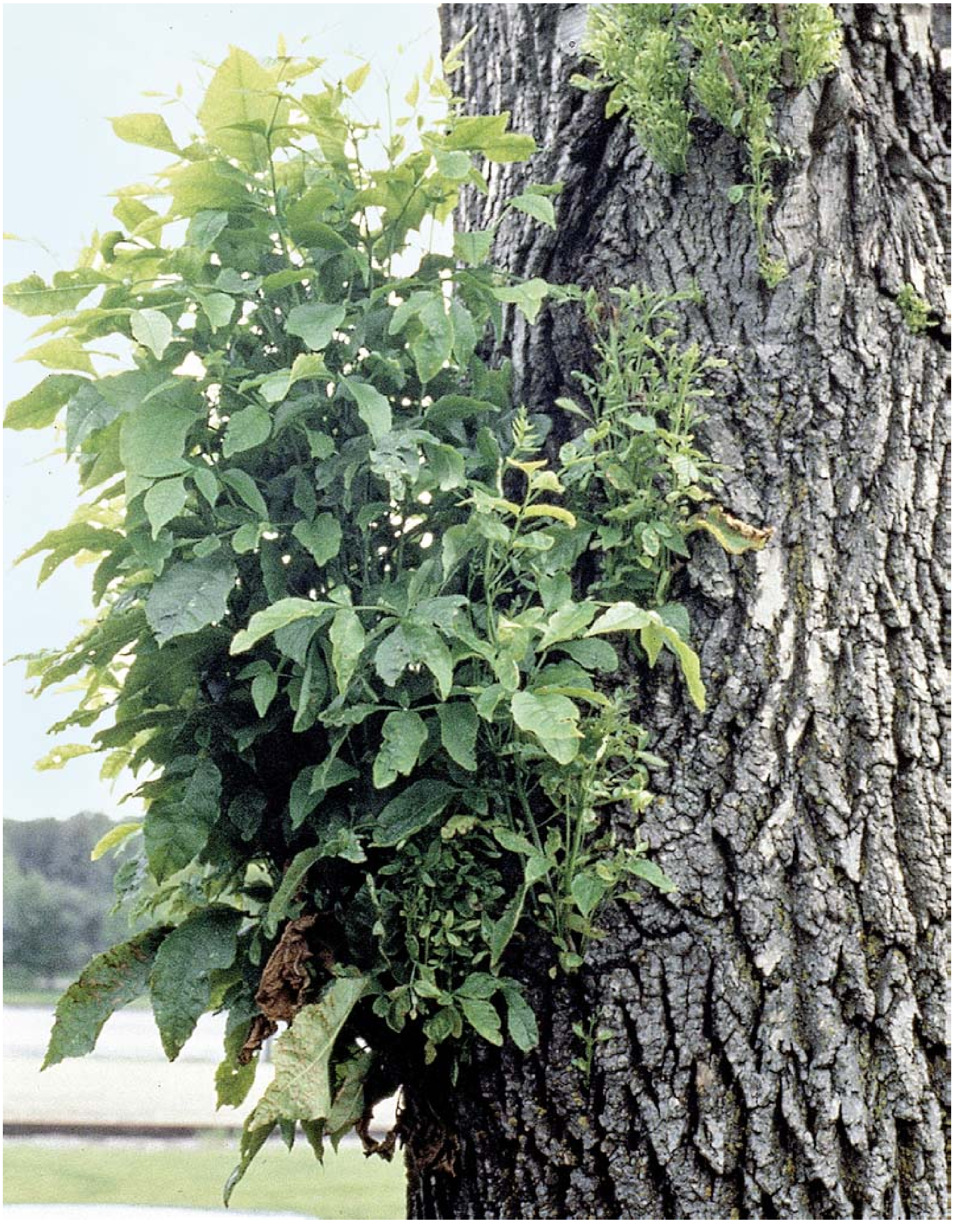


Figure 8.25.
Ash yellows on a green ash tree, caused by a phytoplasma. Note the witches' broom on the trunk.

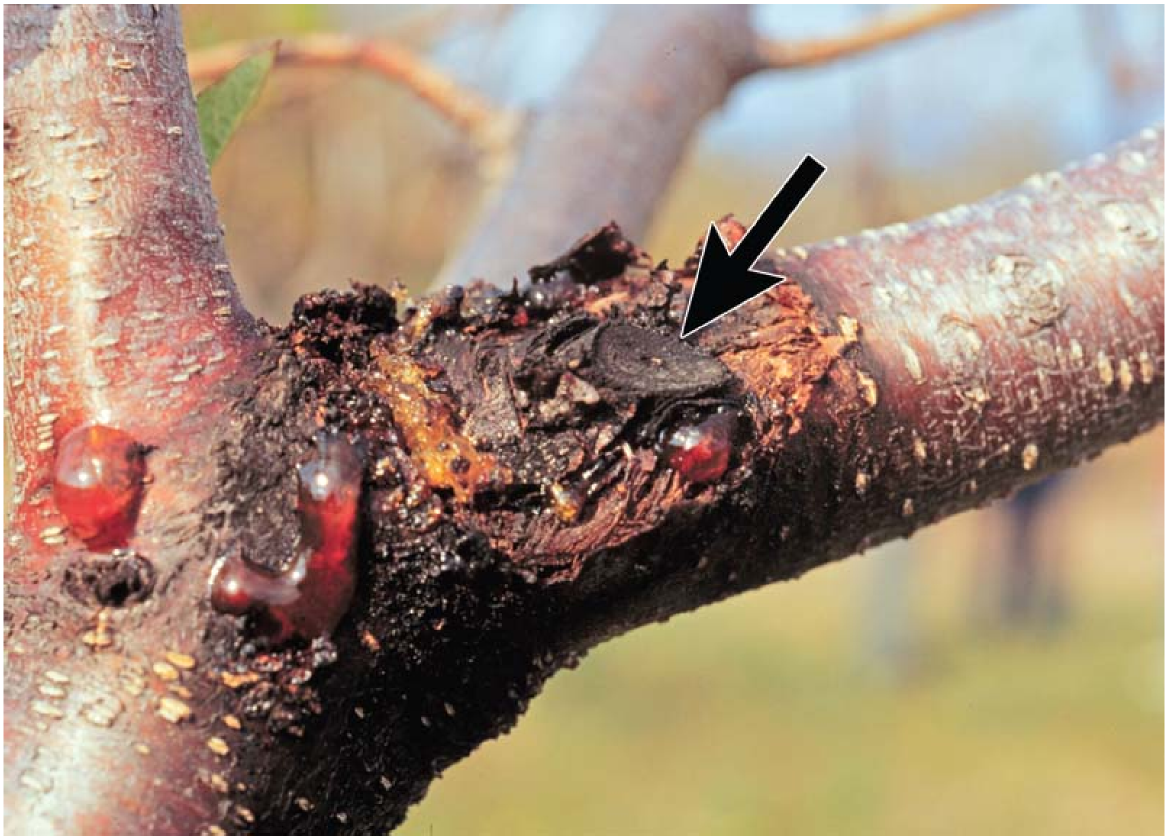


Figure 8.26.
Leucostoma canker is a fungal disease and is one of the most important diseases of stone fruits in the world. Note the amber gum oozing from the infected tissues. A pruning cut (arrow) in the center of the canker served as the infection court.

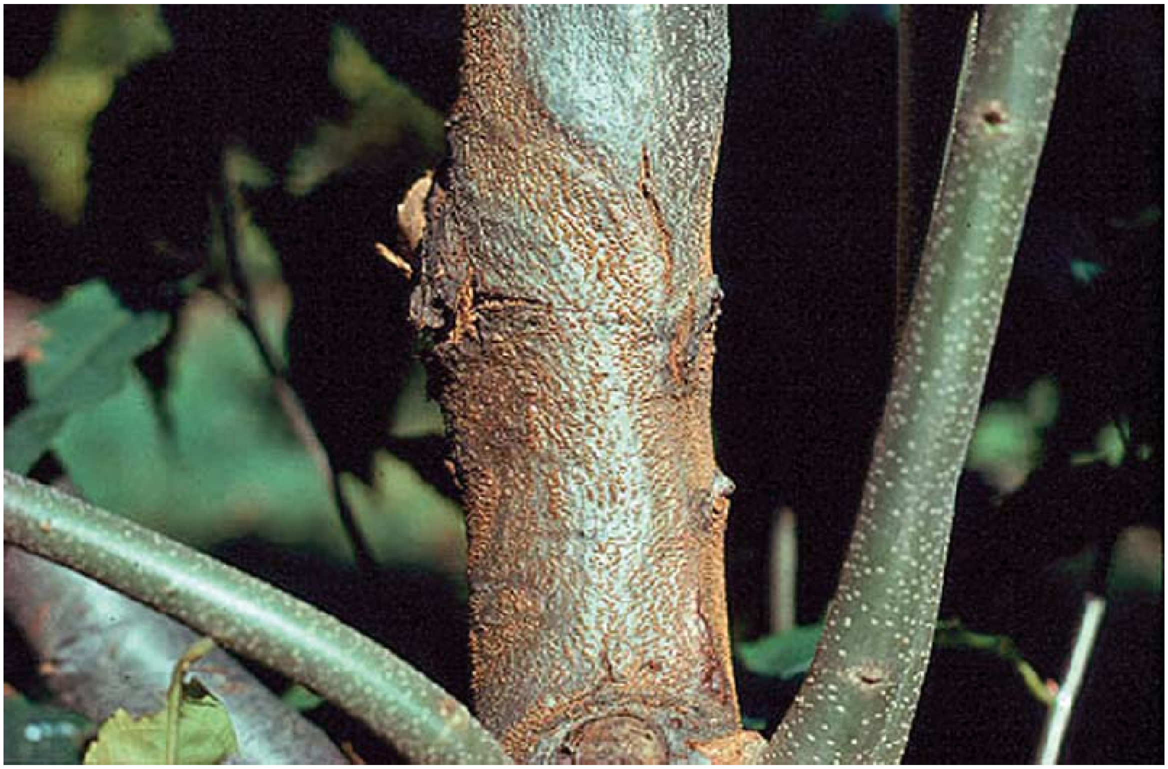


Figure 8.27.
Chestnut blight canker.

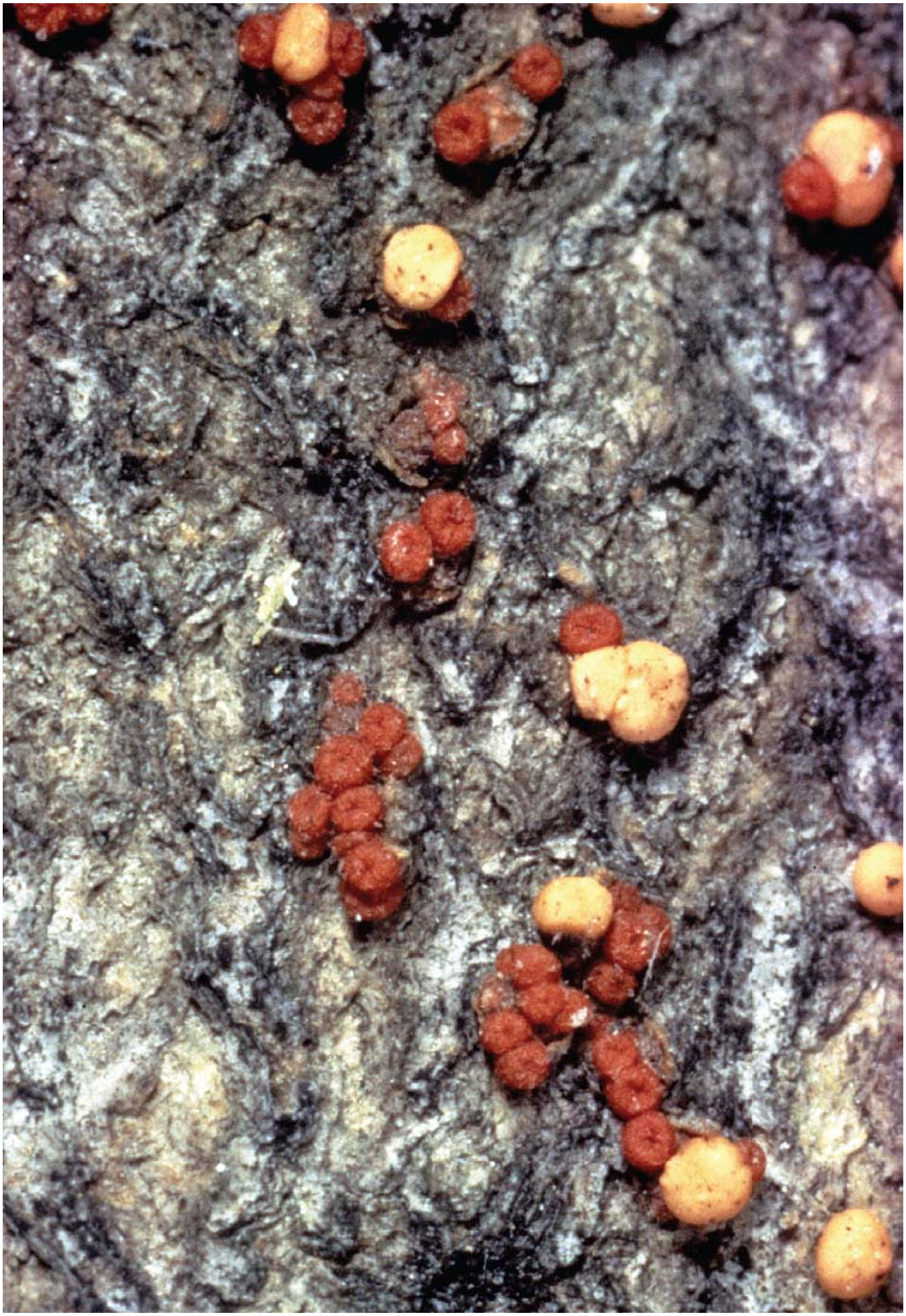


Figure 8.28.
Nectria canker (coral spot) on a hardwood tree. Note the fruiting bodies emerging through the bark.

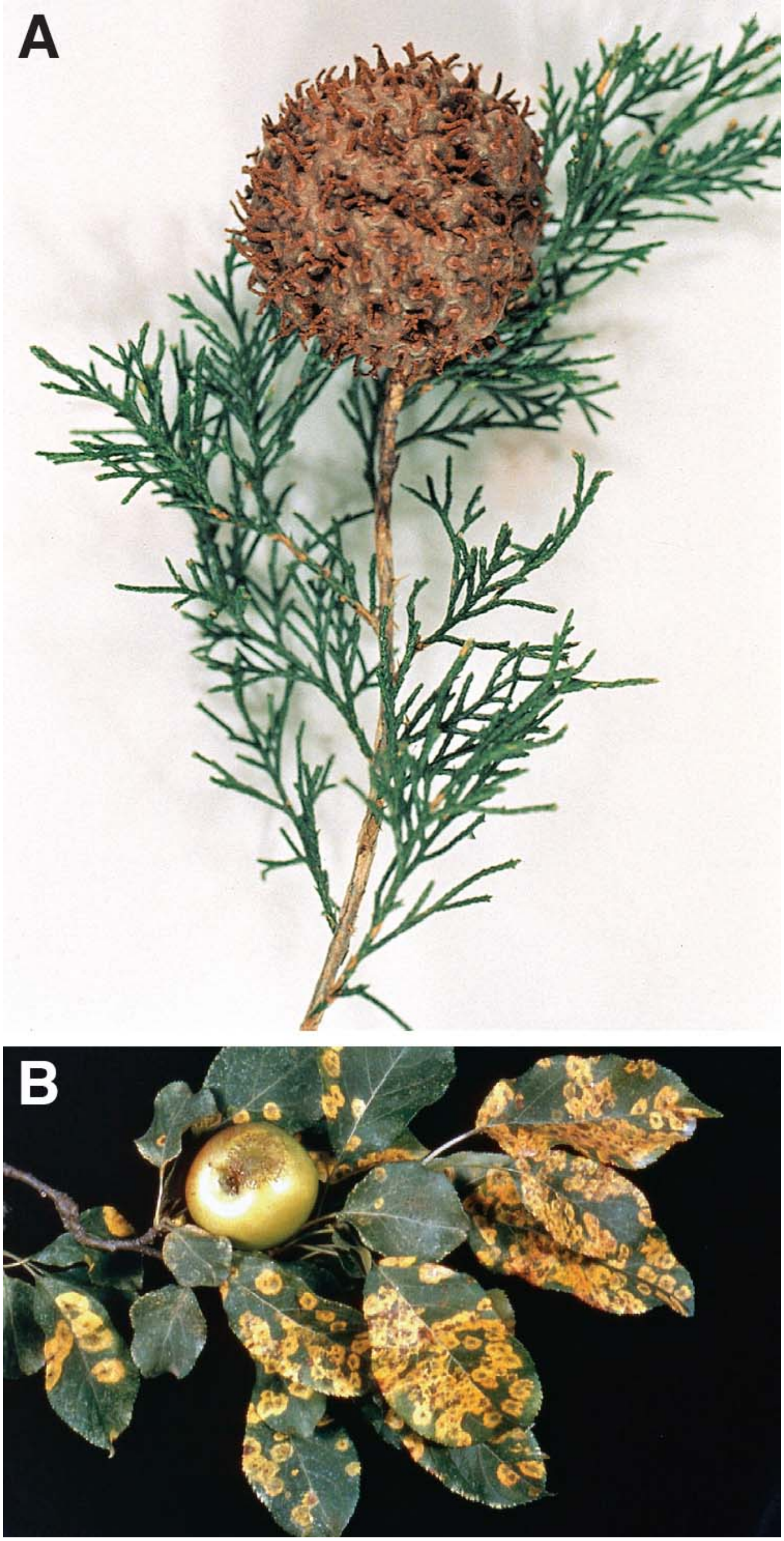

A
B

Figure 8.29
A, Telial gall of cedar-apple rust on red-cedar (juniper). The orange spore horns have withered after basidiospore production. **B,** Cedarapple rust symptoms on apple. The infections were caused by basidiospores produced on the telial gall, resulting in pycnia. Later, aeciospores are produced from aecia on the lower leaf surfaces.

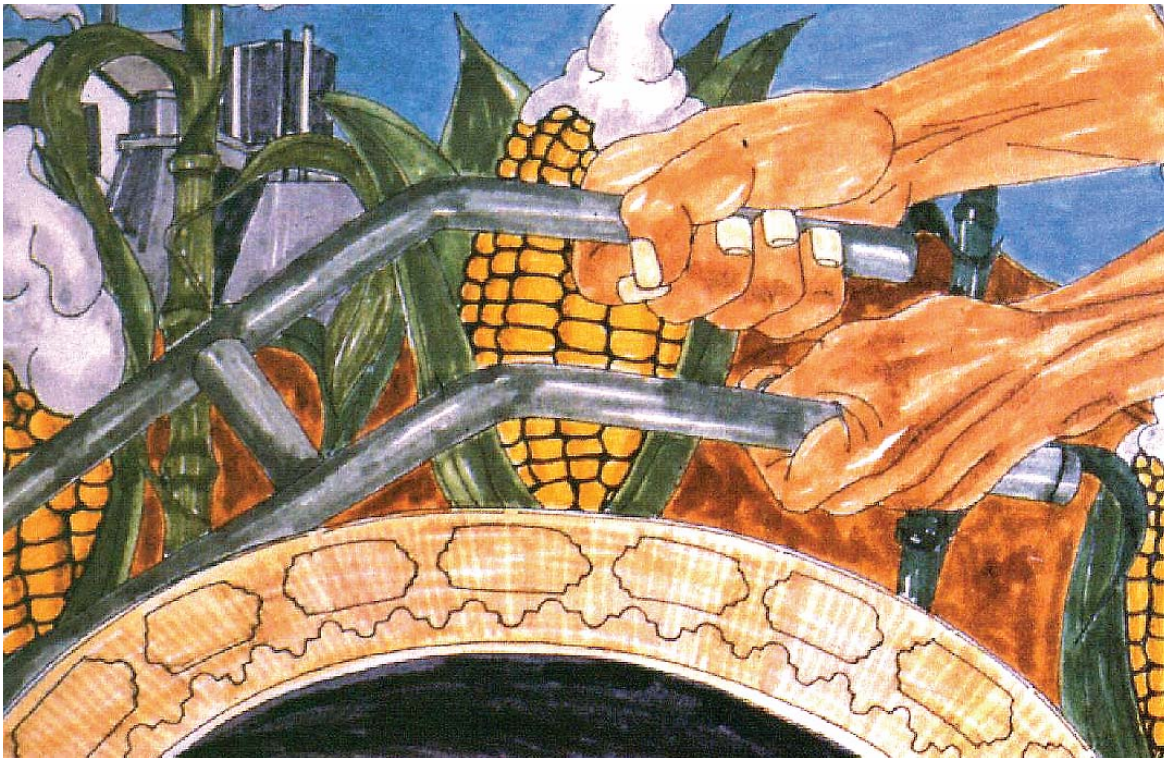


Figure 8.30.
Portion of a mural painted by Guillermo Lourdes in the city of Durango, Mexico, showing the importance of corn in Meso-american culture. Note corn smut on the ears of corn. From a painted copy by Rudy Cruz.

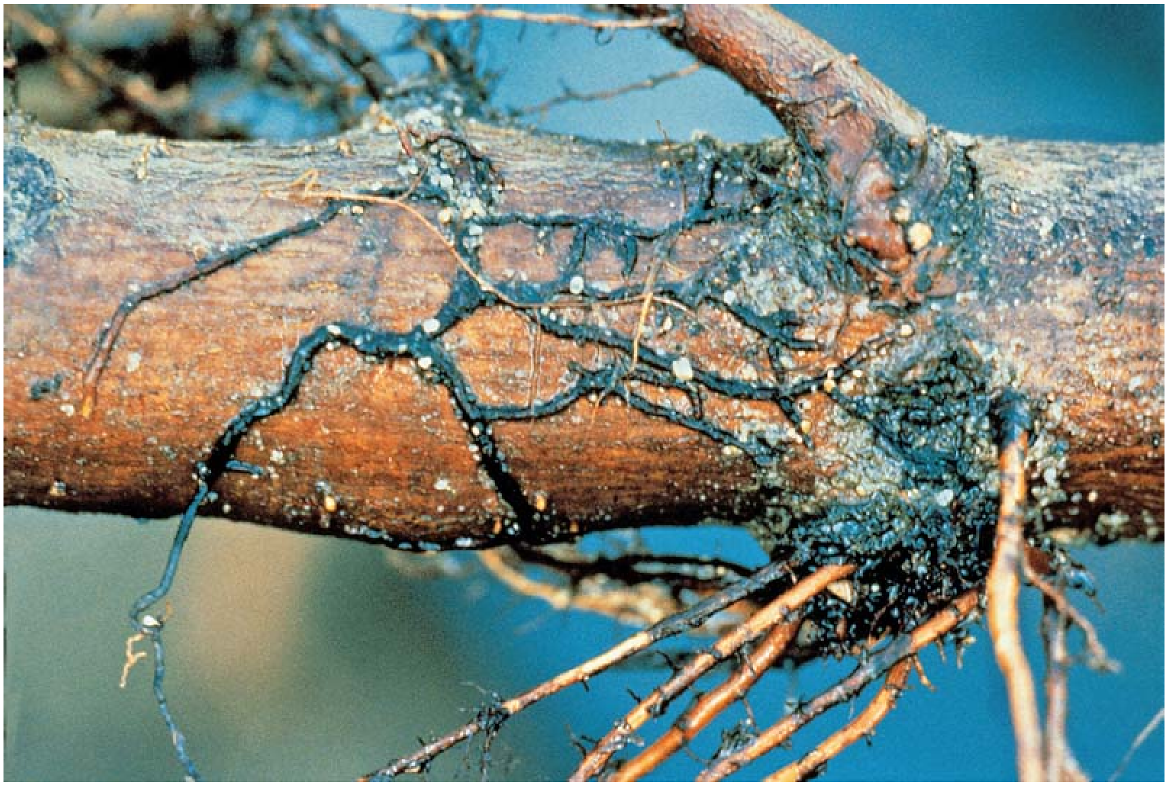


Figure 8.31.
Rhizomorphs (“shoestrings”) of an *Armillaria* sp. adhering to roots of an apple tree.

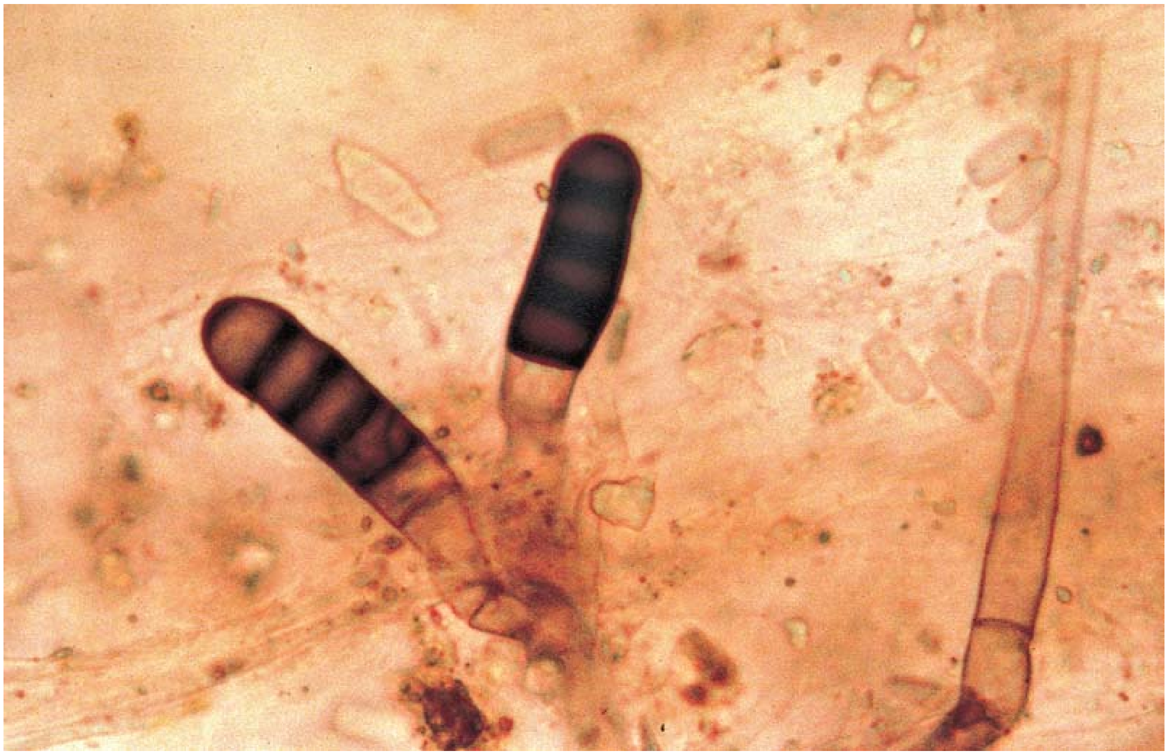


Figure 8.32.
Chlamydospores (left) and endoconidia (right) of *Thielaviopsis basicola*, the black root rot pathogen.

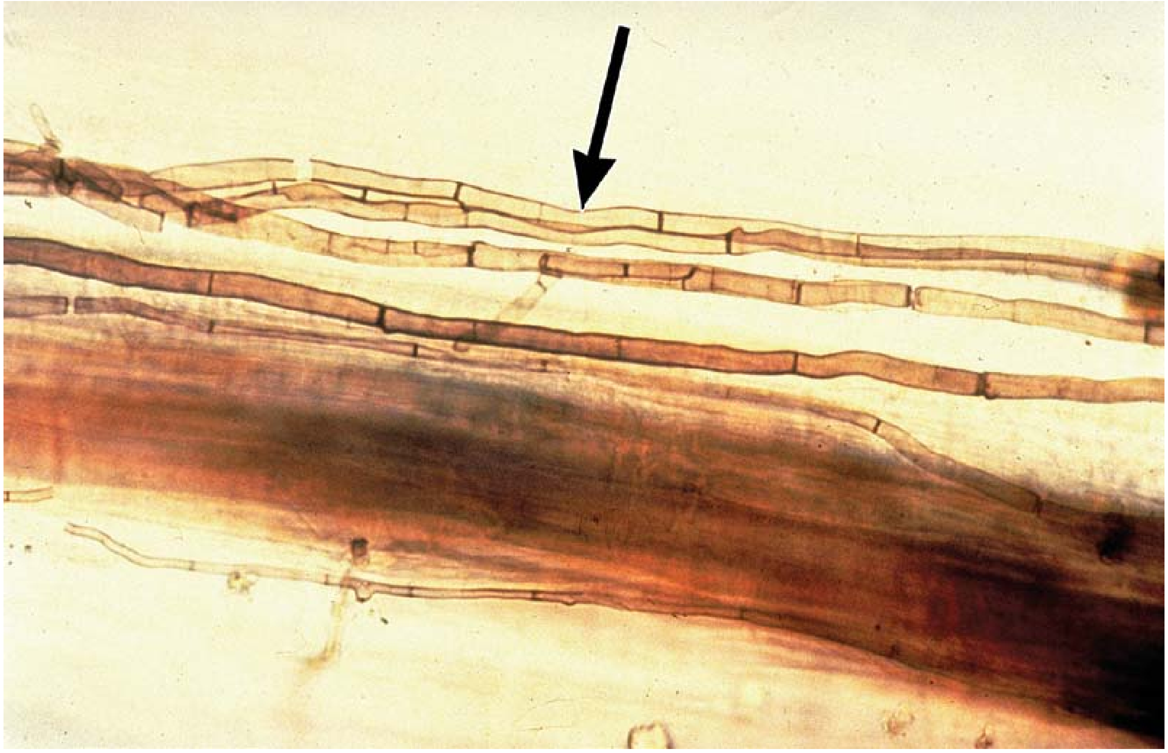


Figure 8.33.
Ectotrophic root-infecting hyphae (arrow), or runner hyphae, on a root of a turfgrass plant.

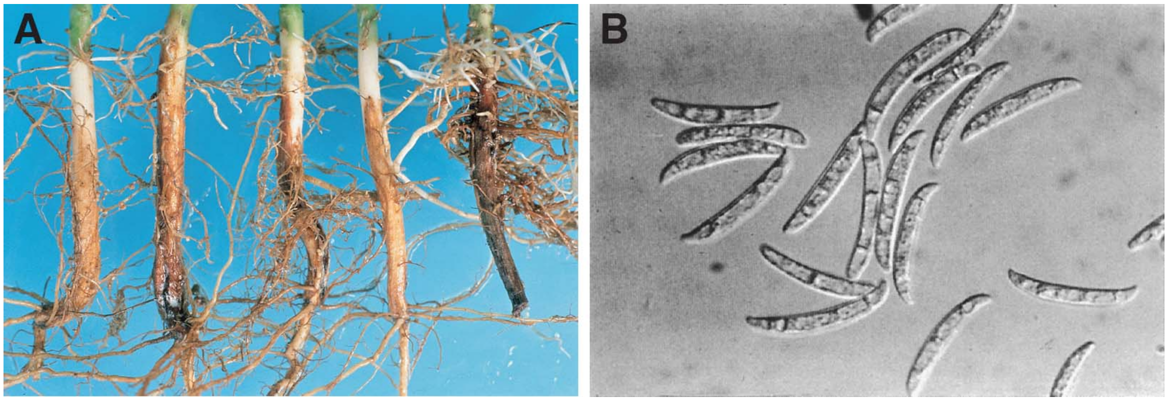
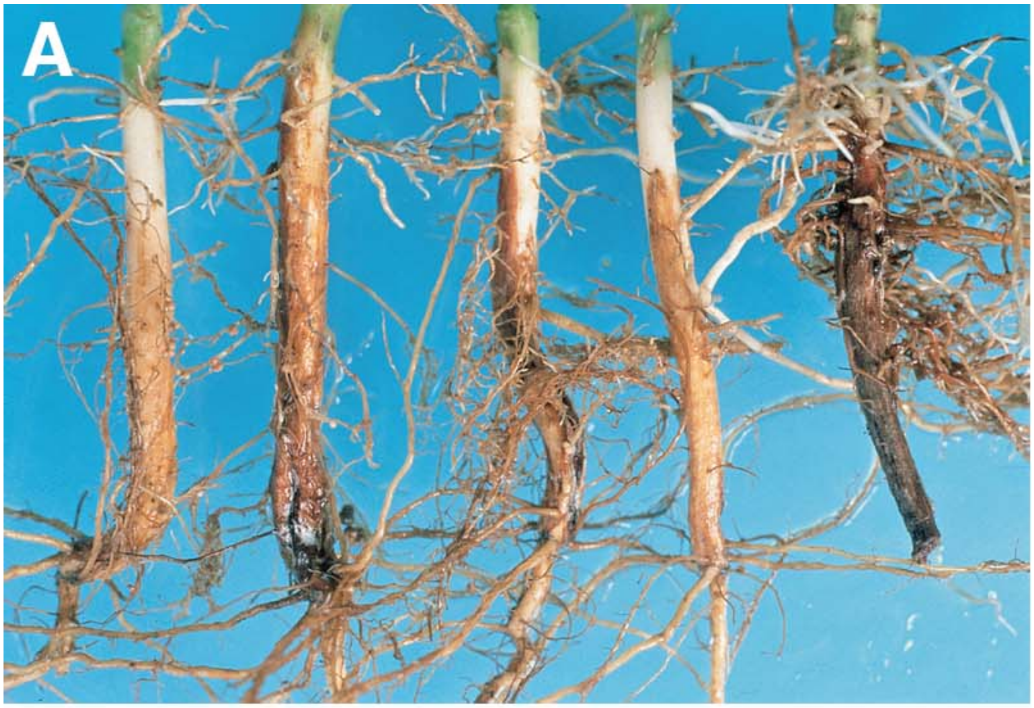


Figure 8.34.
A, Fusarium root rot of bean. **B,** Macroconidia of a *Fusarium* sp., showing the fusoid shape that gives the fungus its name.


A

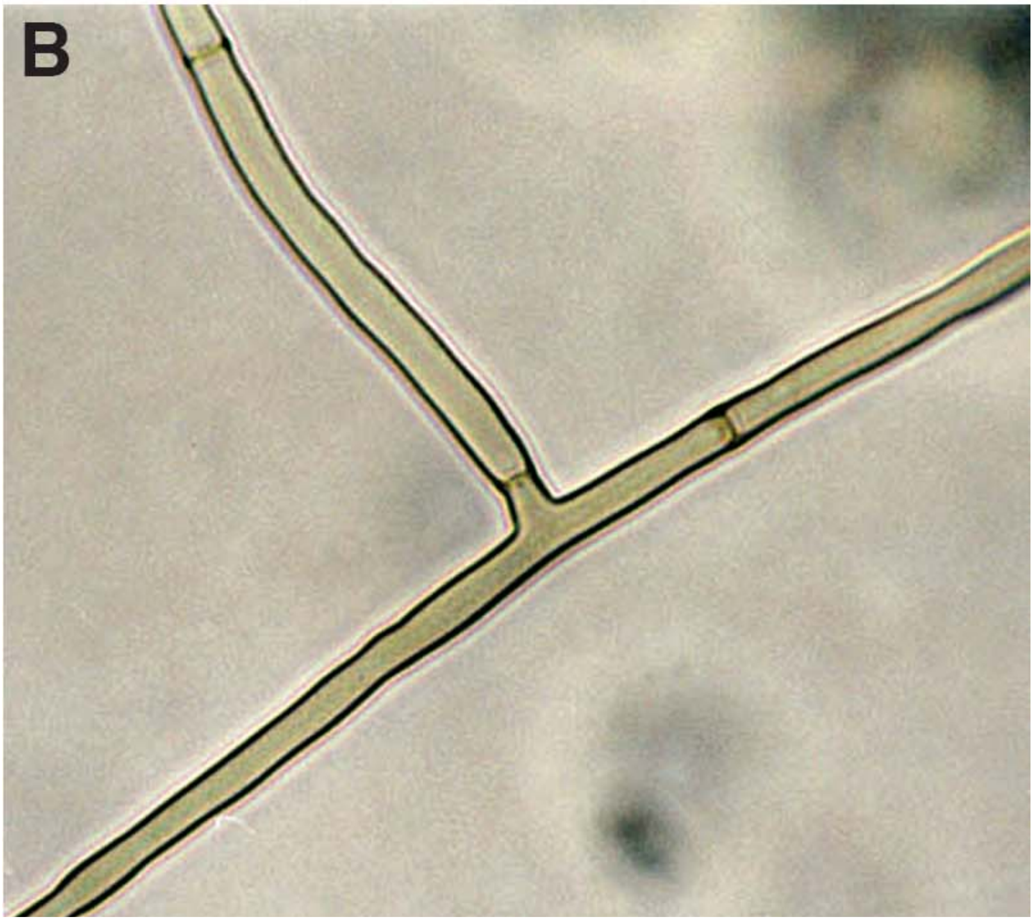

B

Figure 8.35.
A, Sclerotia of *Rhizoctonia* on a potato tuber with black scurf. **B,** Characteristic right-angled branching of *Rhizoctonia* hyphae.

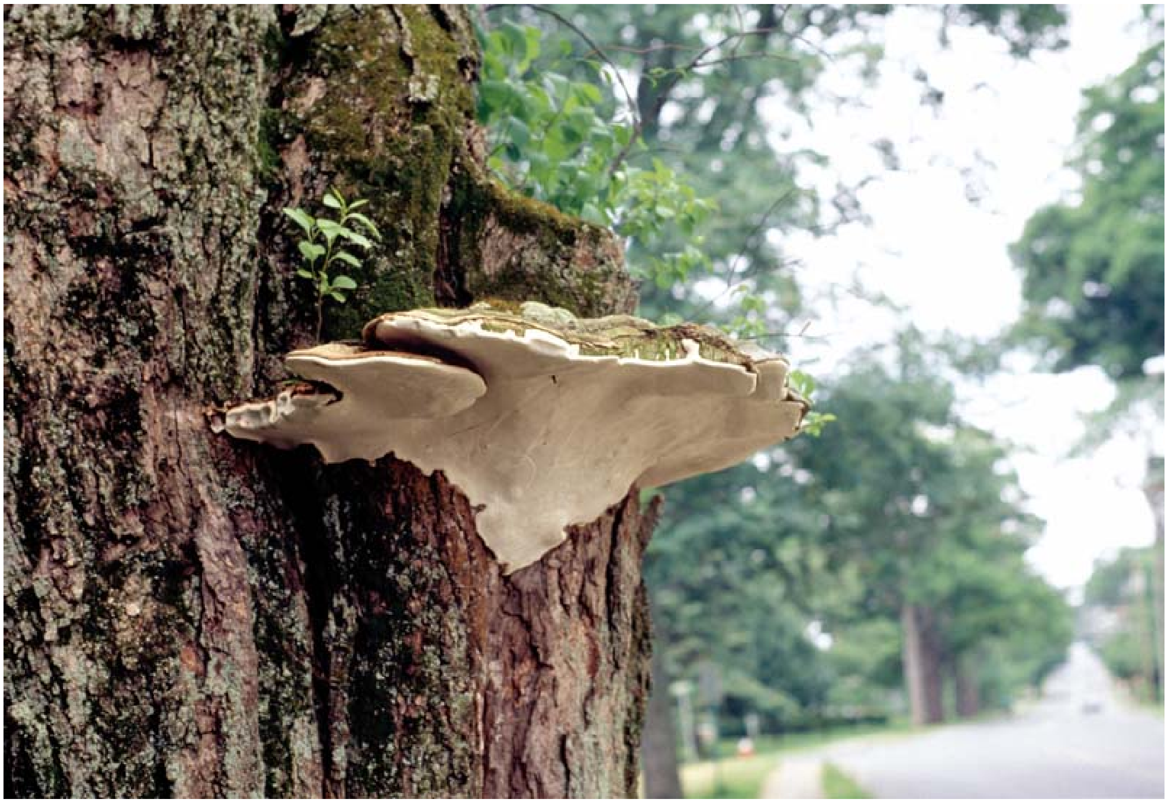


Figure 8.36.
Fruiting body (conk) of a wood decay basidiomycete on the trunk of an infected tree. Basidiospores are produced in the tiny pores on the lower surface. Removal of the conk will not save the tree.

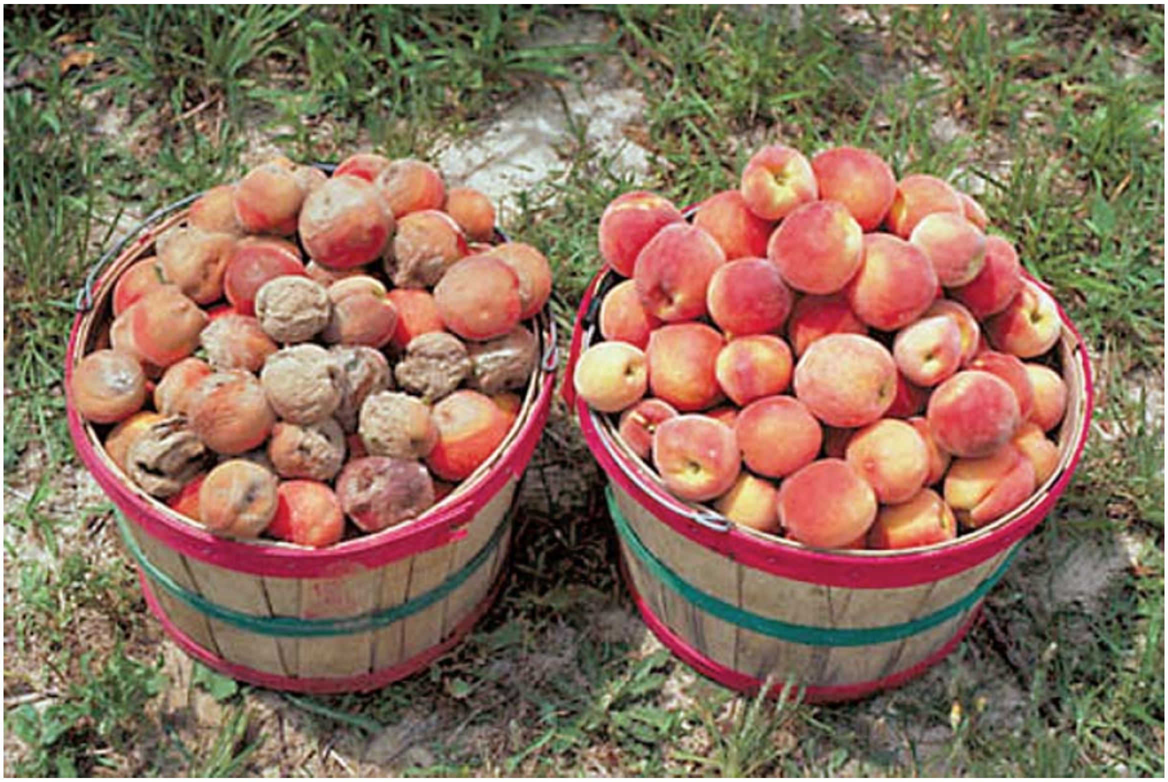


Figure 8.37.
Peaches with brown rot (left), caused by *Monilinia fructicola*.

PLEASE, NO
VOMITOXIN

Figure 8.38.
Humans and other animals can be poisoned by mycotoxins, such as vomitoxin.

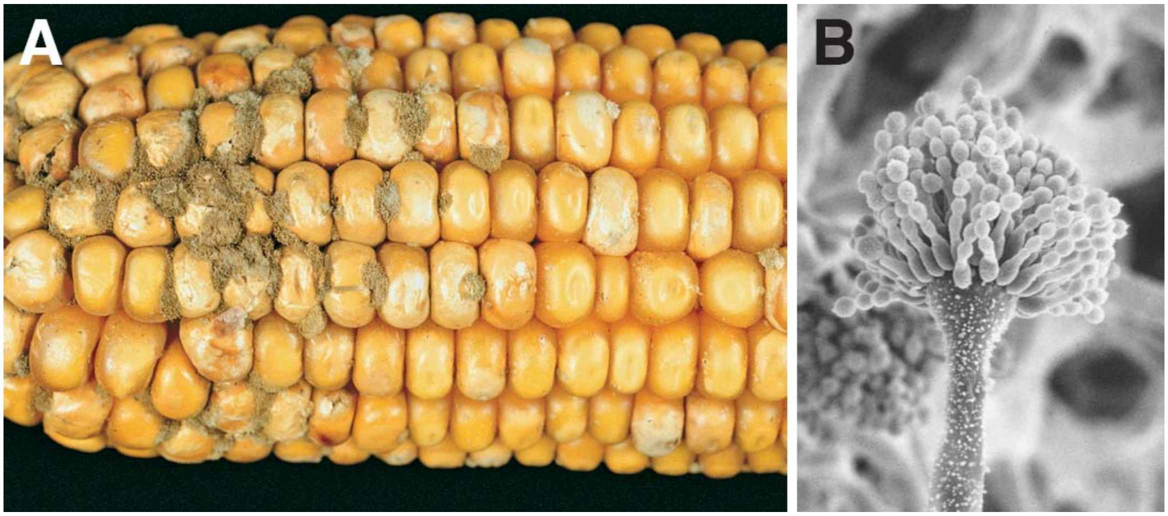


Figure 8.39.
A, Aspergillus ear rot of corn. *Aspergillus flavus*, one fungal species that causes corn ear rot, produces aflatoxin, a dangerous mycotoxin. **B,** Conidiophore of *Aspergillus flavus*. Note the numerous conidia (scanning electron micrograph, ×1,000).

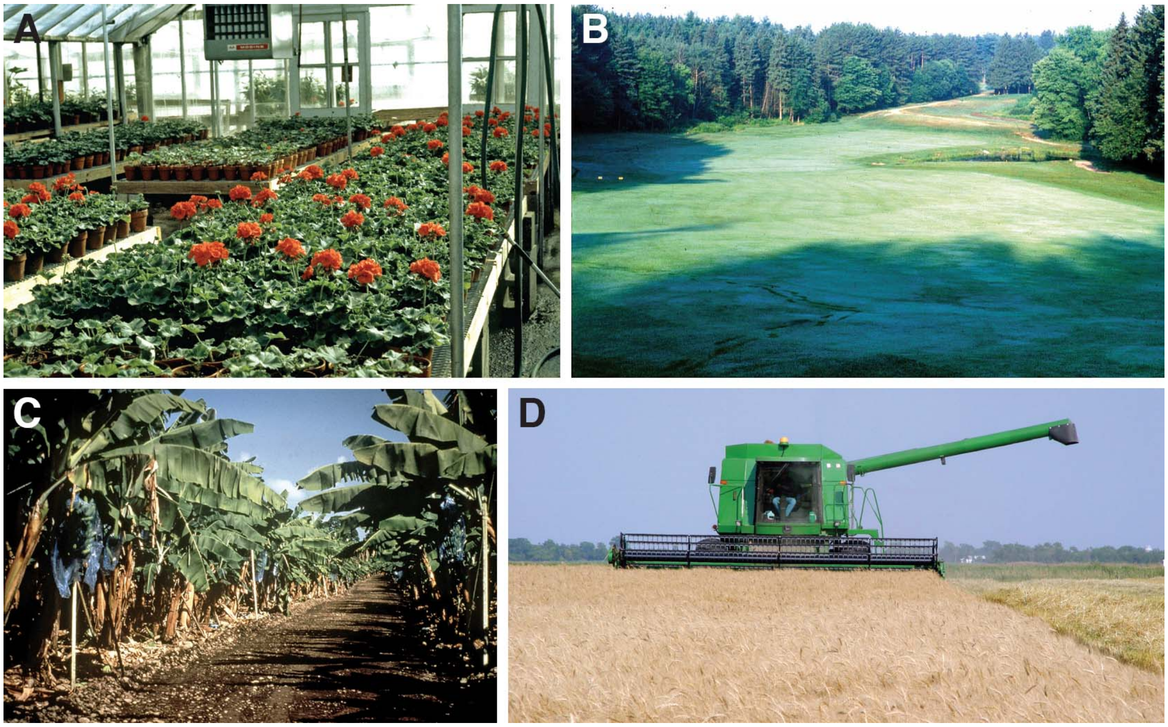


Figure 9.1.
Healthy plants in various agroecosystems. Potential pathogens are not visible but are certainly present. **A,** Geranium crop in a greenhouse. **B,** Turfgrass on a golf course fairway. **C,** Bananas on a plantation in Puerto Rico. **D,** Wheat harvest in the United States.

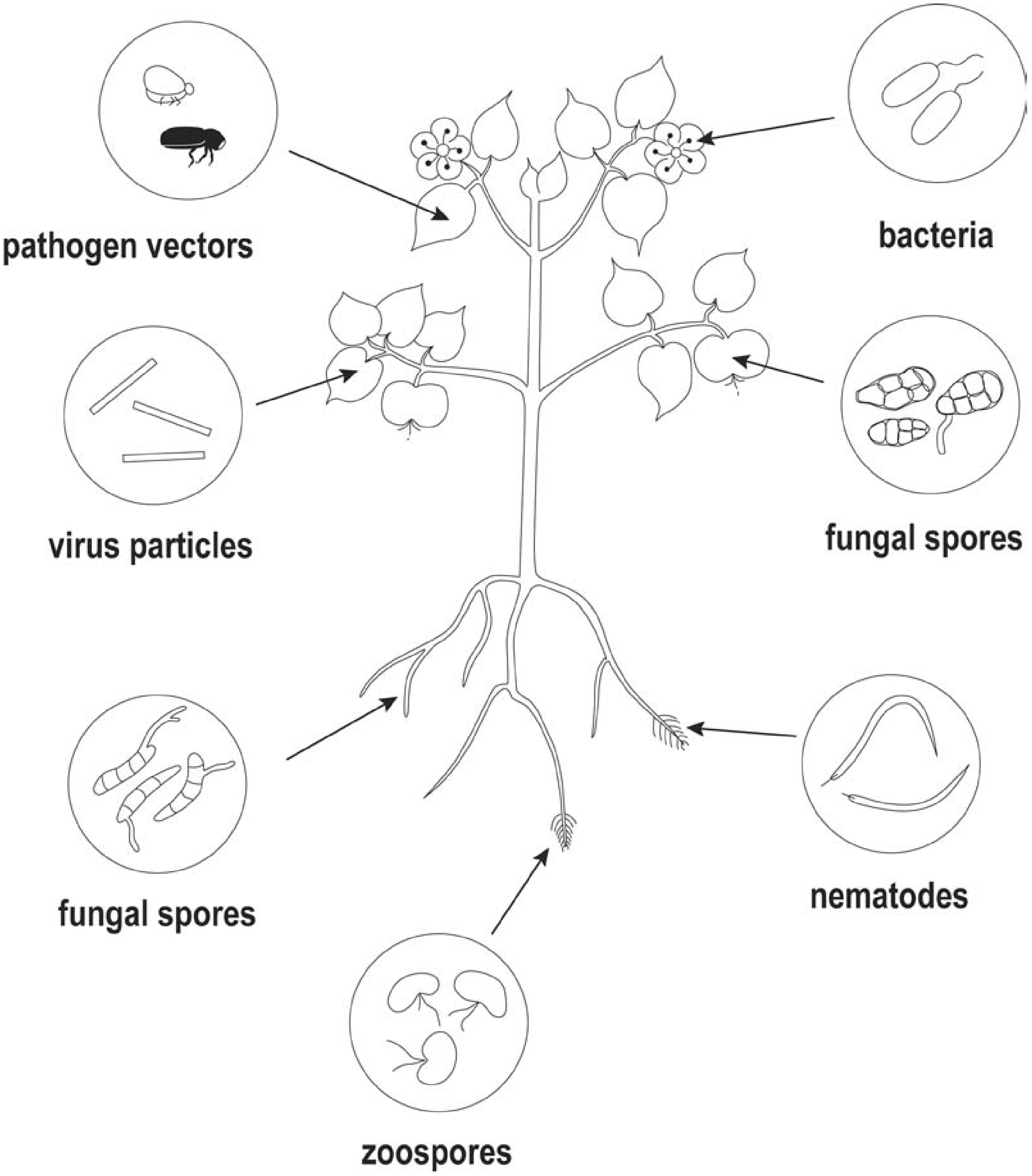

pathogen vectors
bacteria
virus particles
fungal spores
fungal spores
nematodes
zoospores

Figure 9.2.

Plants are exposed to a variety of pathogens.

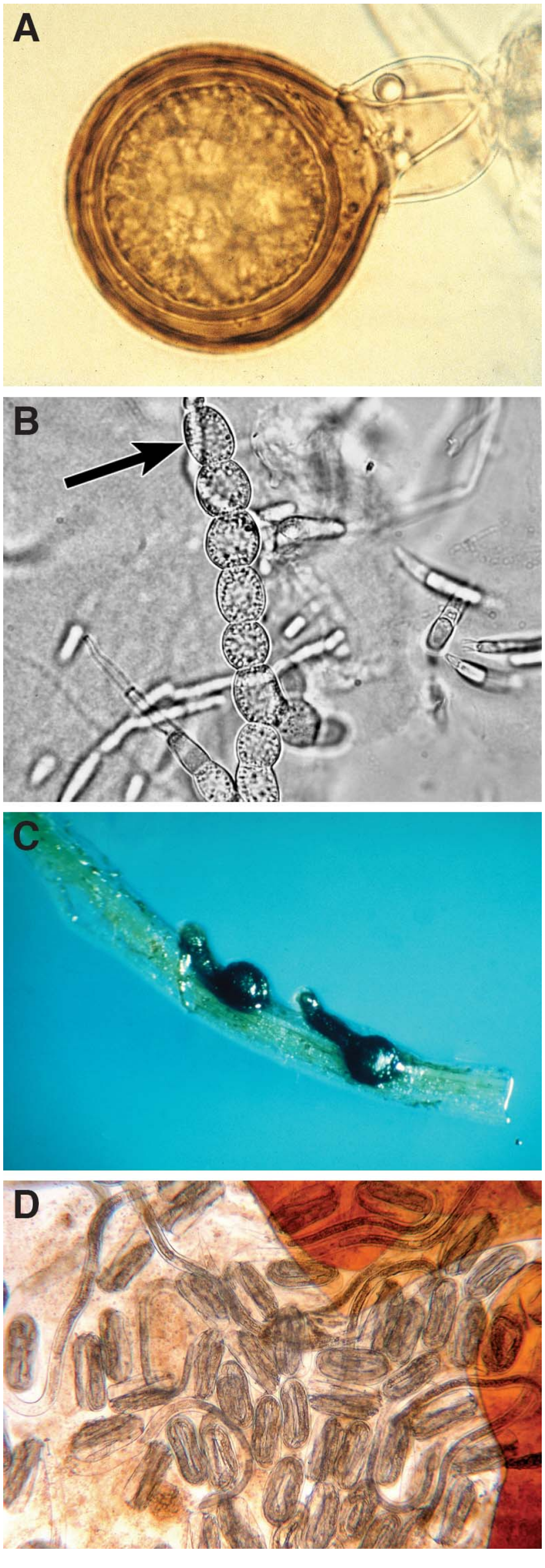

A
B
C
D

Figure 9.3.
Examples of pathogen survival structures. **A,** Thick-walled oospore of *Phytophthora cinnamomi*. **B,** Chlamydospores (arrow) of *Fusarium sporotrichioides.* **C,** Melanized perithecia of *Gaeuman-nomyces graminis* var. *avenae* (take-all root rot). **D,** Nematode cyst opened to expose eggs and second-stage juveniles.

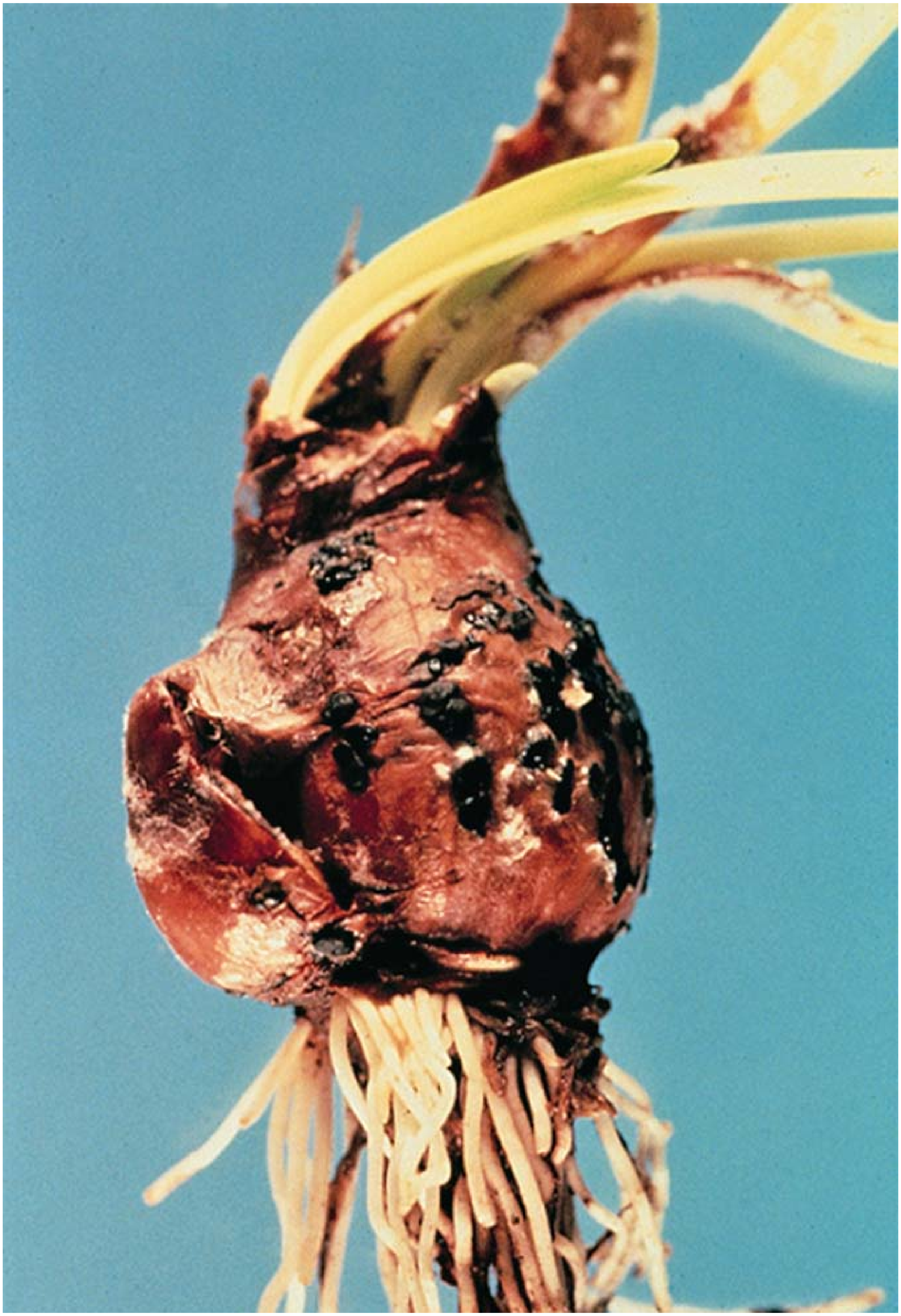


Figure 9.4.
Sclerotia of *Botrytis* sp. (gray mold) and other fungal pathogens can contaminate bulbs and other propagative parts, leading to unintentional spread of the disease.

Figure 9.5.
Cronartium ribicola, which causes white pine blister rust, survives in blister rust cankers.

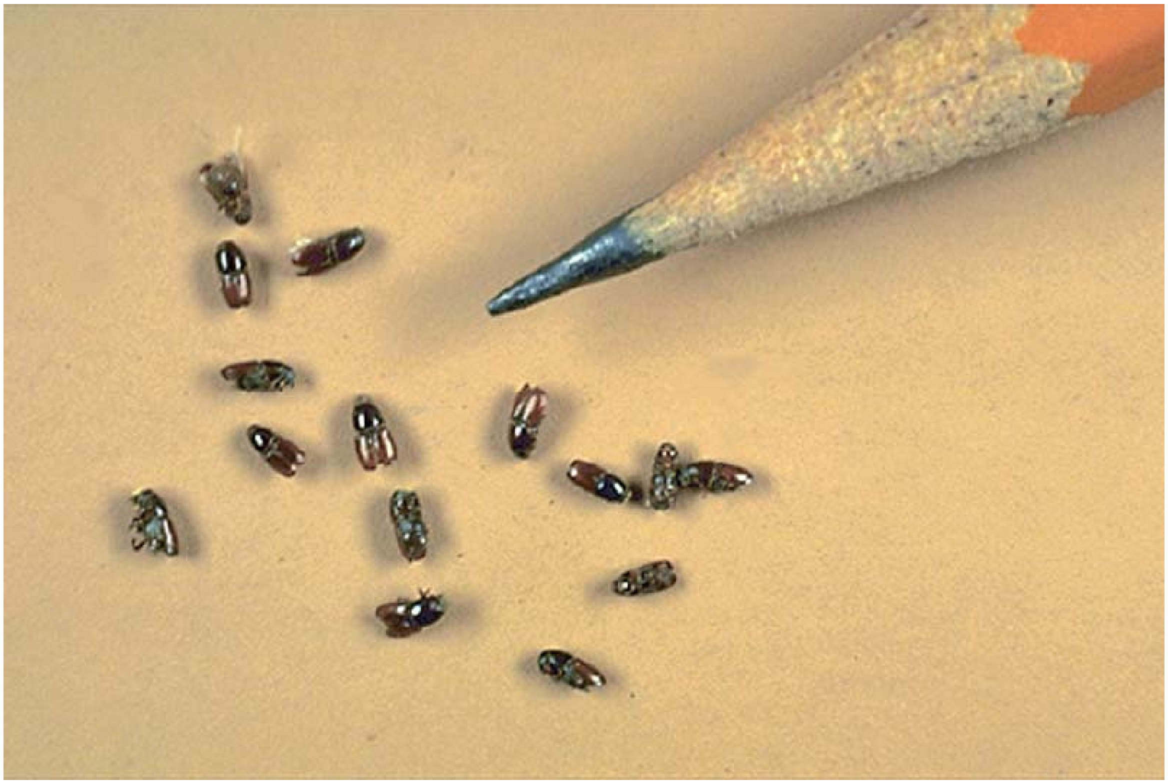


Figure 9.6.
Elm bark beetles survive under the bark of dying elm trees, along with the fungal pathogens that cause Dutch elm disease.

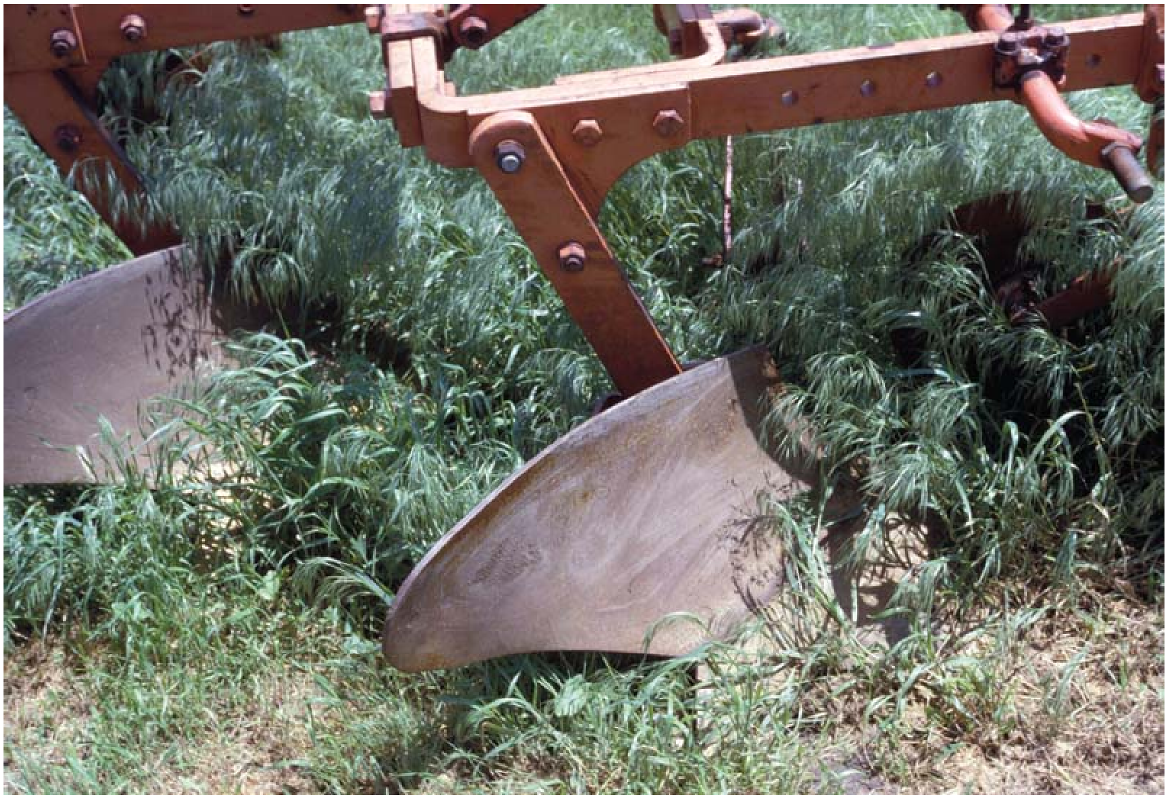


Figure 9.7.
Tillage with a moldboard plow that turns stubble into the soil for degradation. The survival of pathogens is reduced when soil is tilled (plowed) to break up and bury infested plant debris.

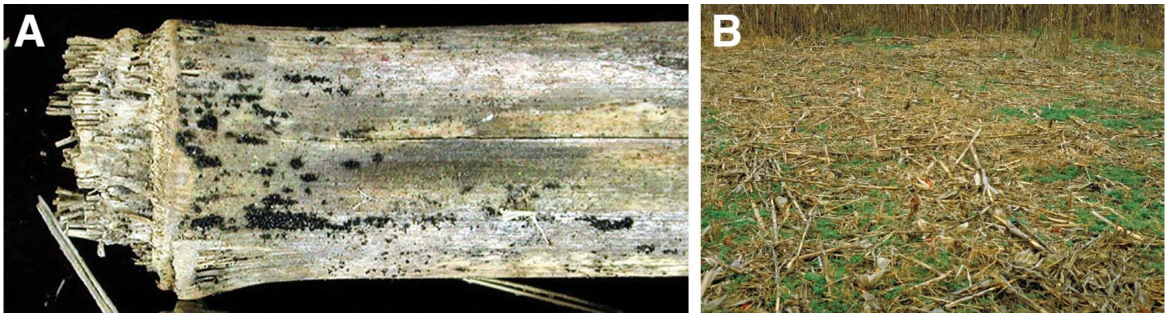


Figure 9.8.

Pathogens surviving in plant debris. **A,** Perithecia of the Fusarium head blight pathogen, *Gibberella zeae* (anamorph: *Fusarium graminearum*), forming on the surface of infested plant debris. **B,** Corn debris infested by the gray leaf spot pathogen, *Cercospora zeae-maydis*.

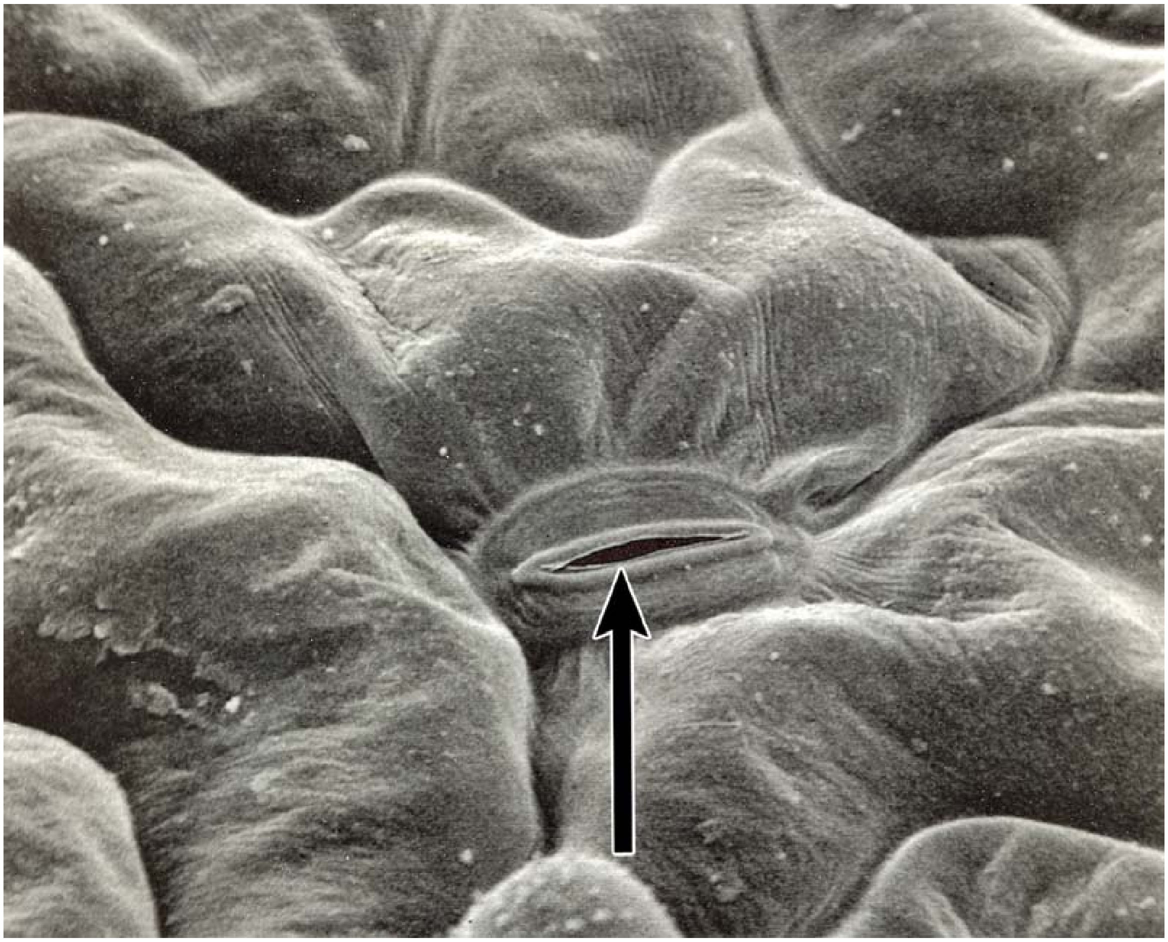


Figure 9.9.
Stoma (arrow) in a leaf surface, a common infection court for foliar plant pathogens (shown in a scanning electron micrograph).

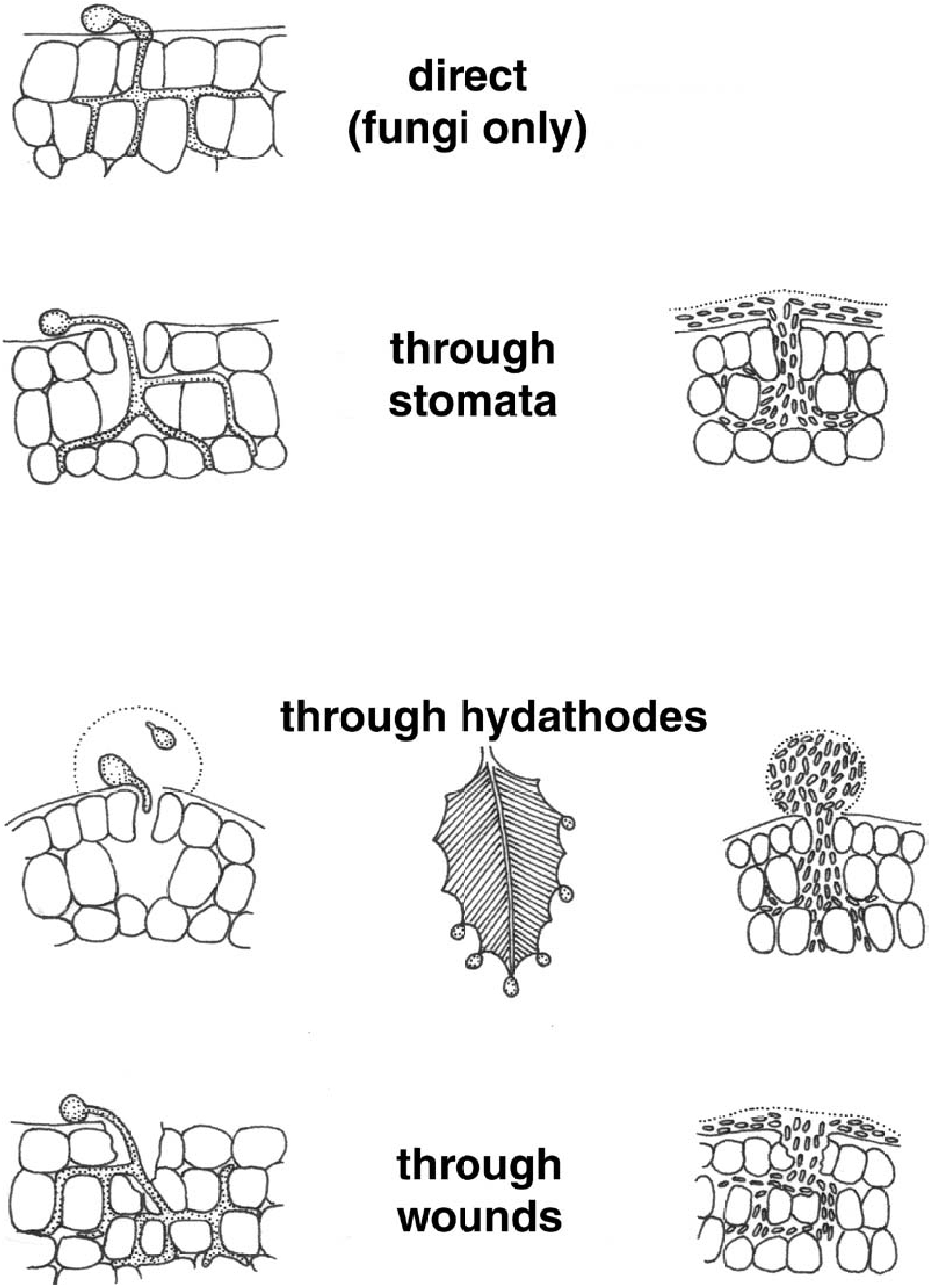

direct
(fungi only)
through
stomata
through hydathodes
through
wounds

Figure 9.10.

Direct and indirect penetration of leaves by fungi (left) and bacteria (right).

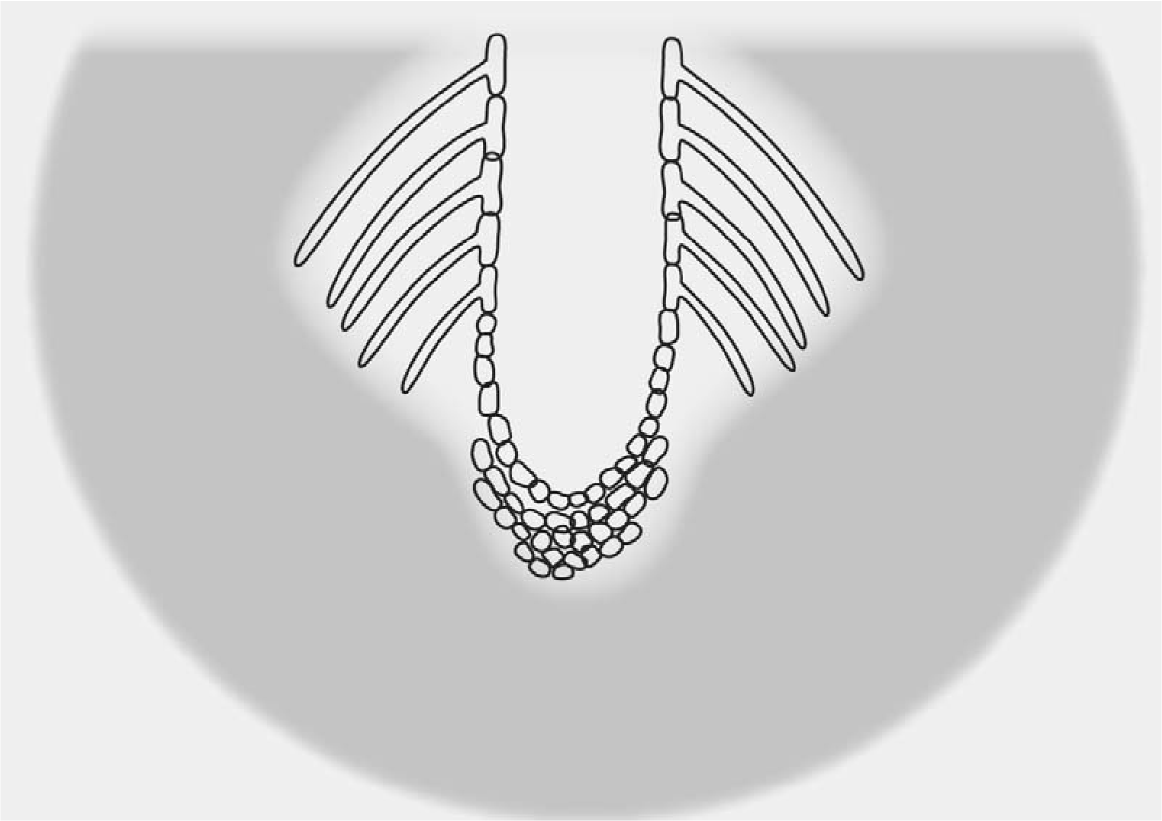


Figure 9.11.
A plant root and its rhizosphere (white area), the zone influenced by root exudates.

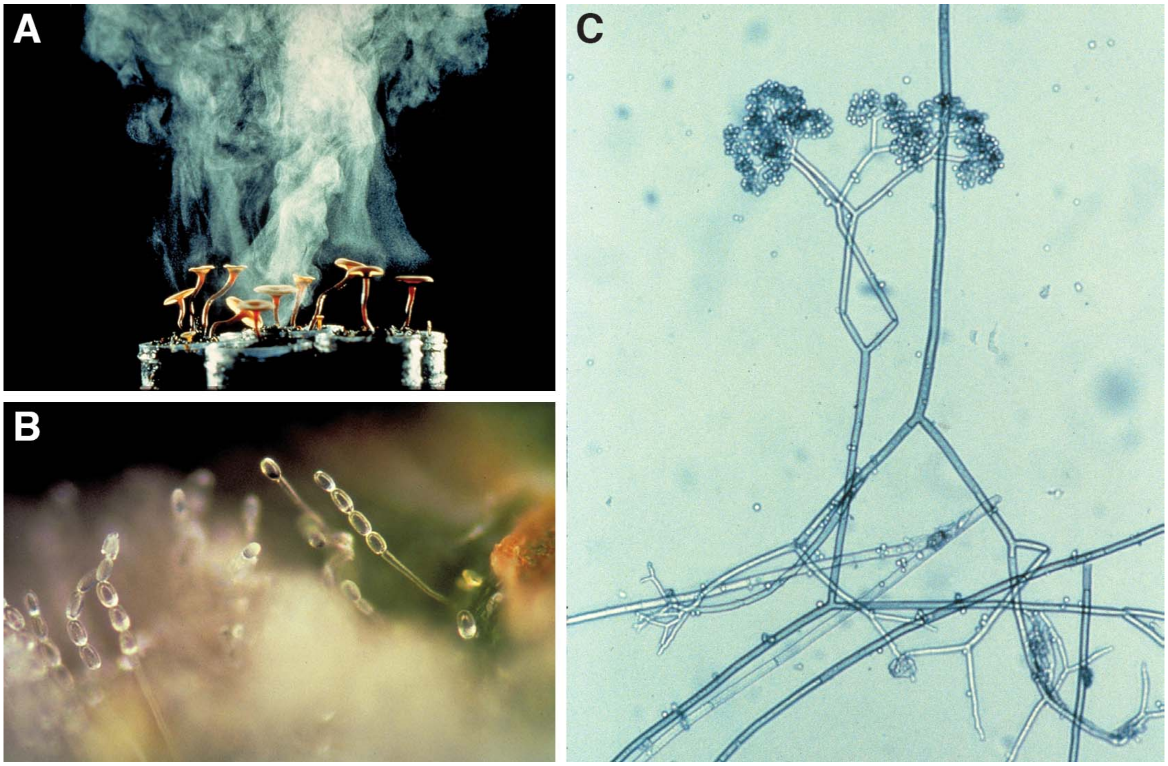


Figure 9.12.
Air dispersal of fungal spores. **A,** Forcible discharge of ascospores (resembling smoke) from the apothecia (fruiting bodies) of *Sclerotinia sclerotiorum* (white mold). **B,** Conidia of a powdery mildew fungus that have formed in chains to reach air currents. **C,** Conidia and conidiophore of *Botrytis cinerea* (gray mold). The conidiophore raises the conidia above the surface of the infected plant, plant debris, or soil to aid in air dispersal.

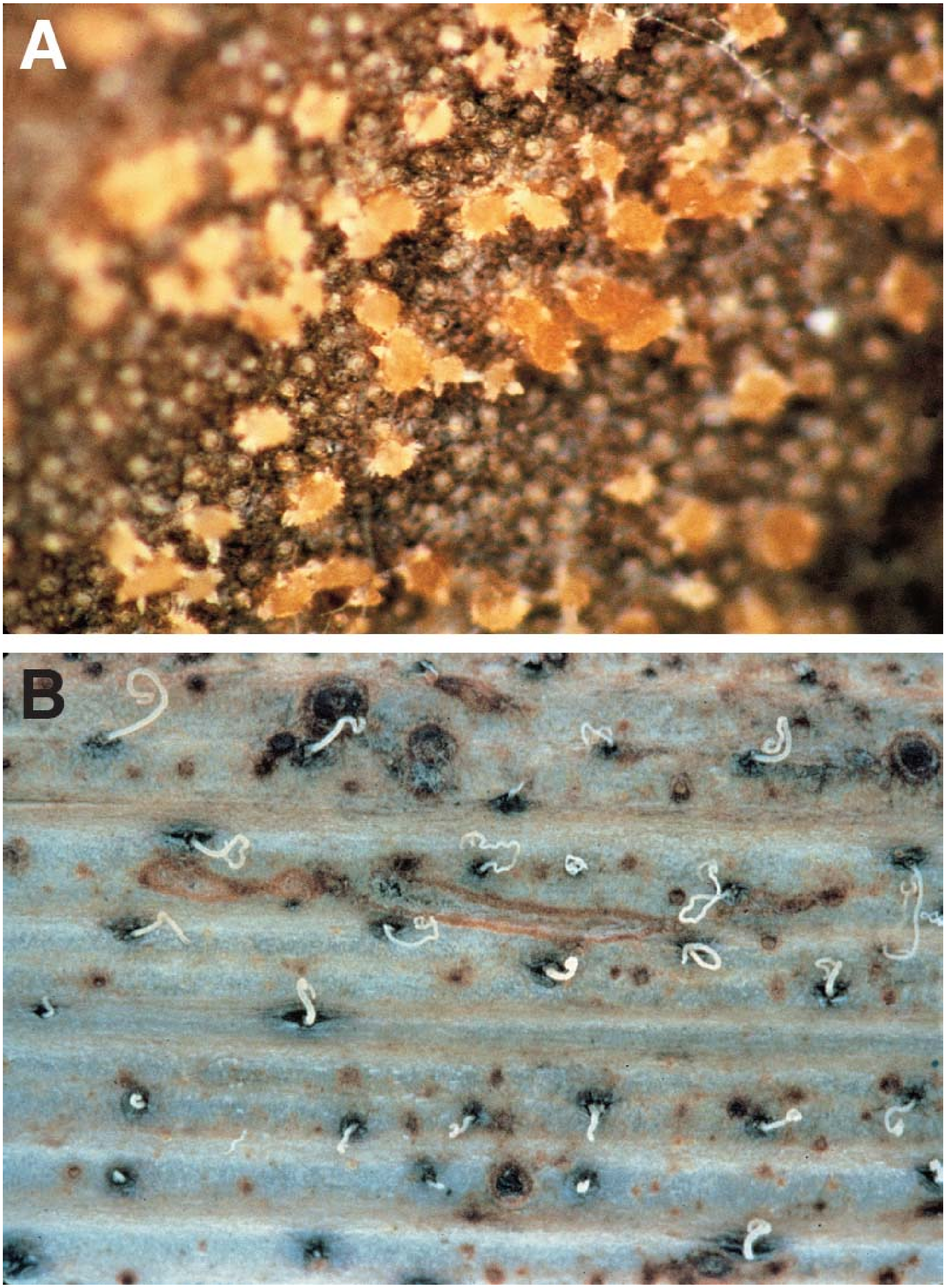


Figure 9.13.

Sticky fungal spores may be dispersed by splashing water, insects, machinery, or tools. **A,** Masses of sticky spores. **B,** Pycnidia exuding conidia in tendril-like masses of slimy spores (cirrhi) on the surface of an infected stem.

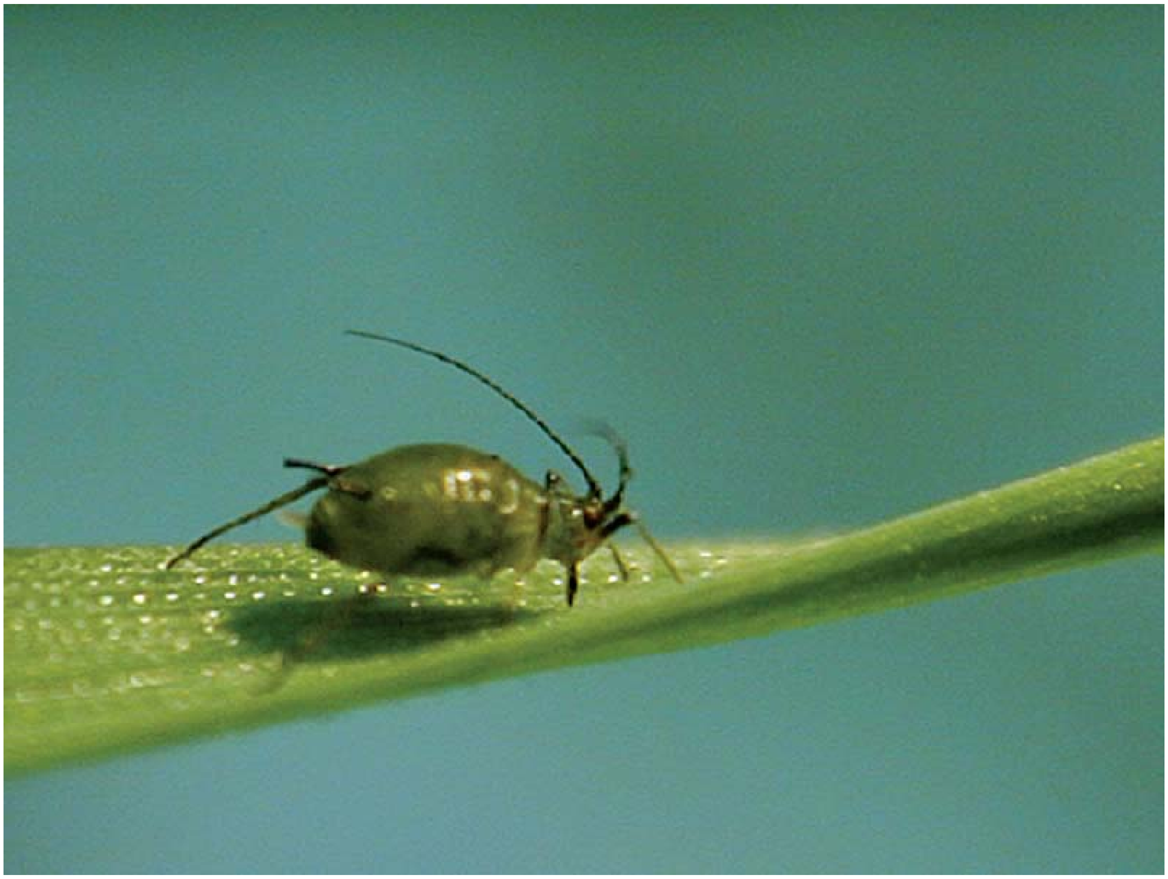


Figure 9.14.
When an aphid feeds on a leaf, it becomes a potential vector for a plant virus.

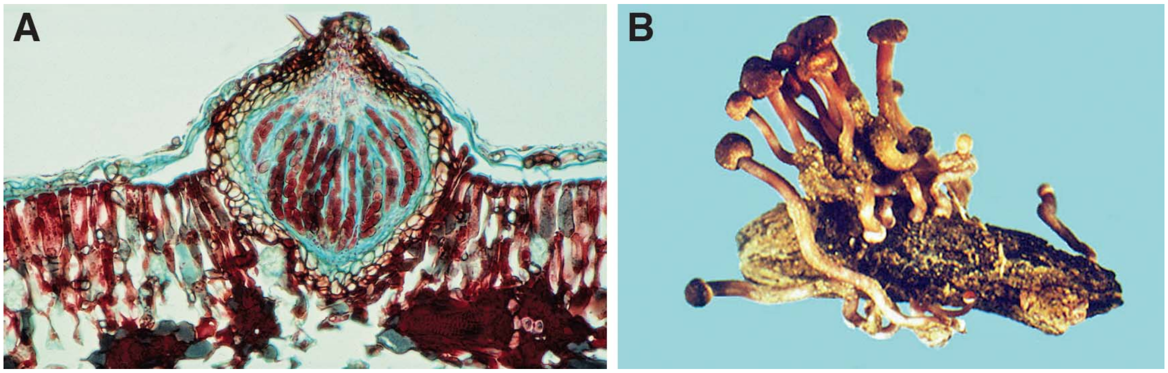


Figure 9.15.
Phenological synchrony allows pathogens to produce inoculum at the time that host plants are susceptible to infection. **A,** A pseudothecium of the apple scab pathogen, *Venturia inaequalis*, from which ascospores are forcibly discharged during the period when apple leaves can be infected. **B,** A germinated sclerotium (ergot) (*Claviceps purpurea*) with ascostromata from which ascospores are forcibly discharged at the time that the host grass is flowering.

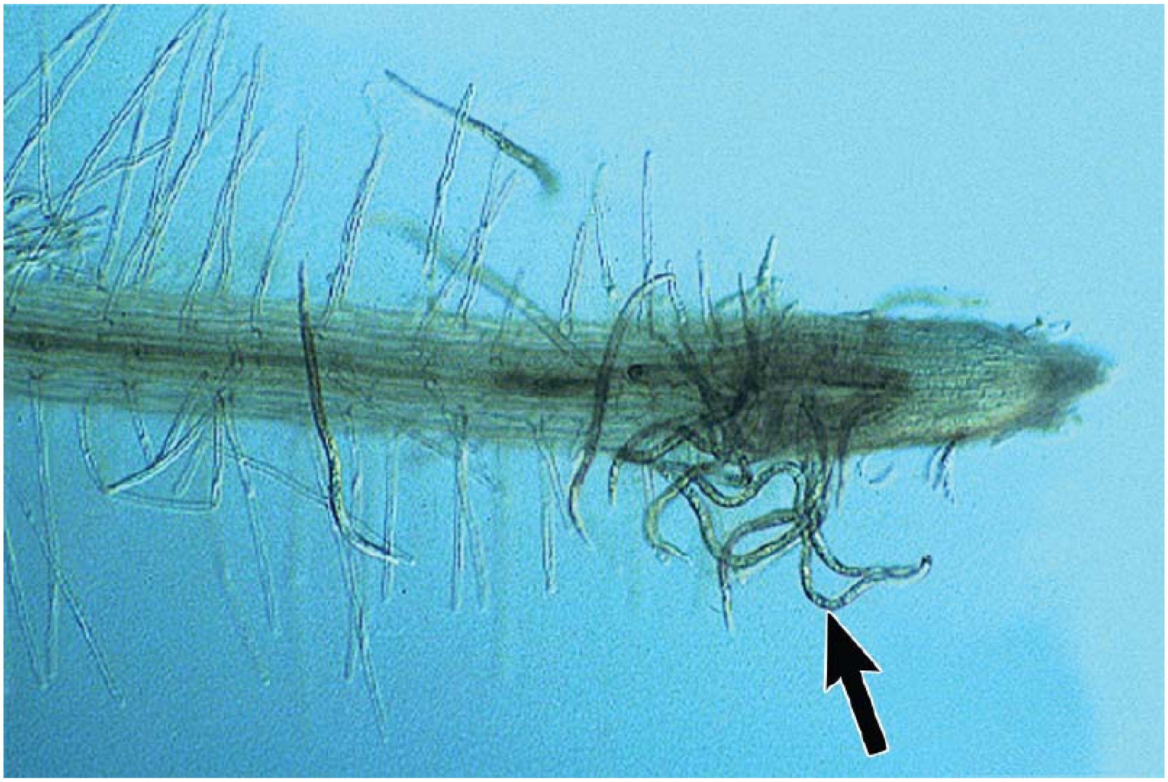


Figure 9.16.
Nematodes (arrow) in the root hair zone. Chemotaxis is the mechanism by which they find this zone.

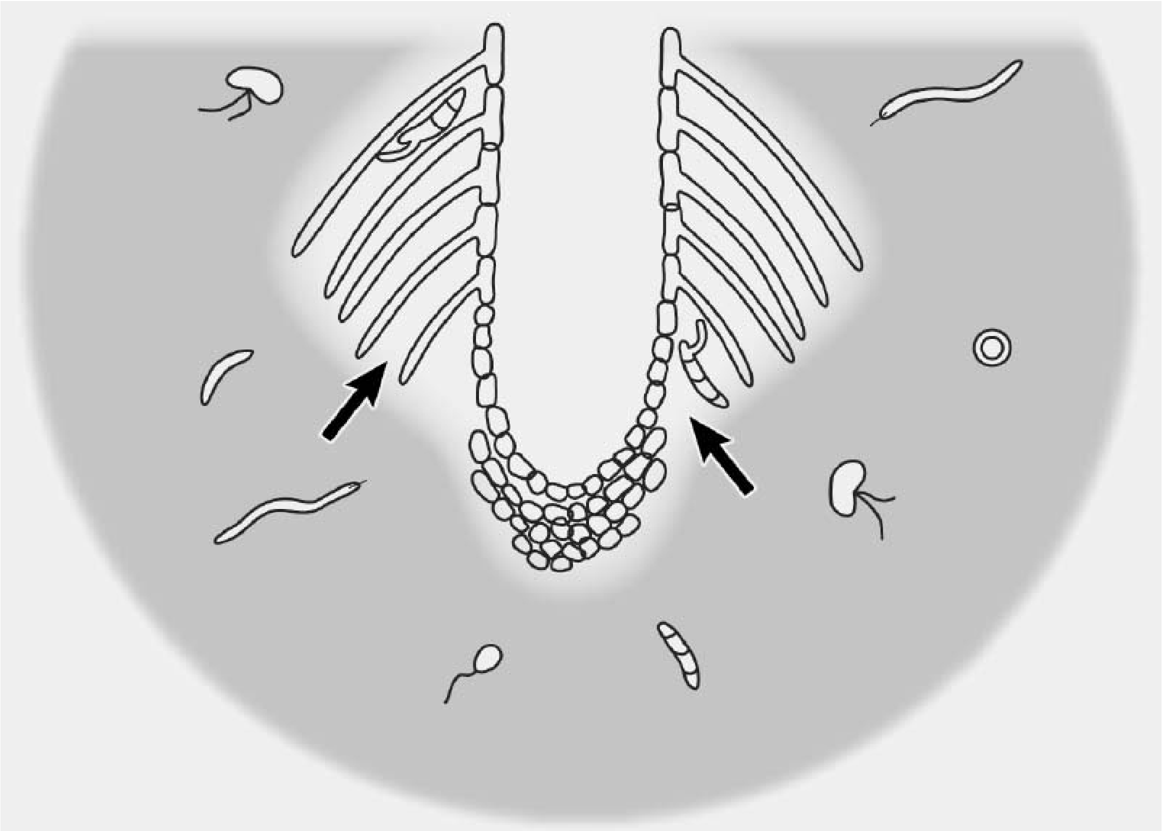


Figure 9.17.

Pathogen activity near the rhizosphere. Note that fungistasis prevents germination of spores outside the rhizosphere. Nematodes and zoospores are attracted to the rhizosphere.

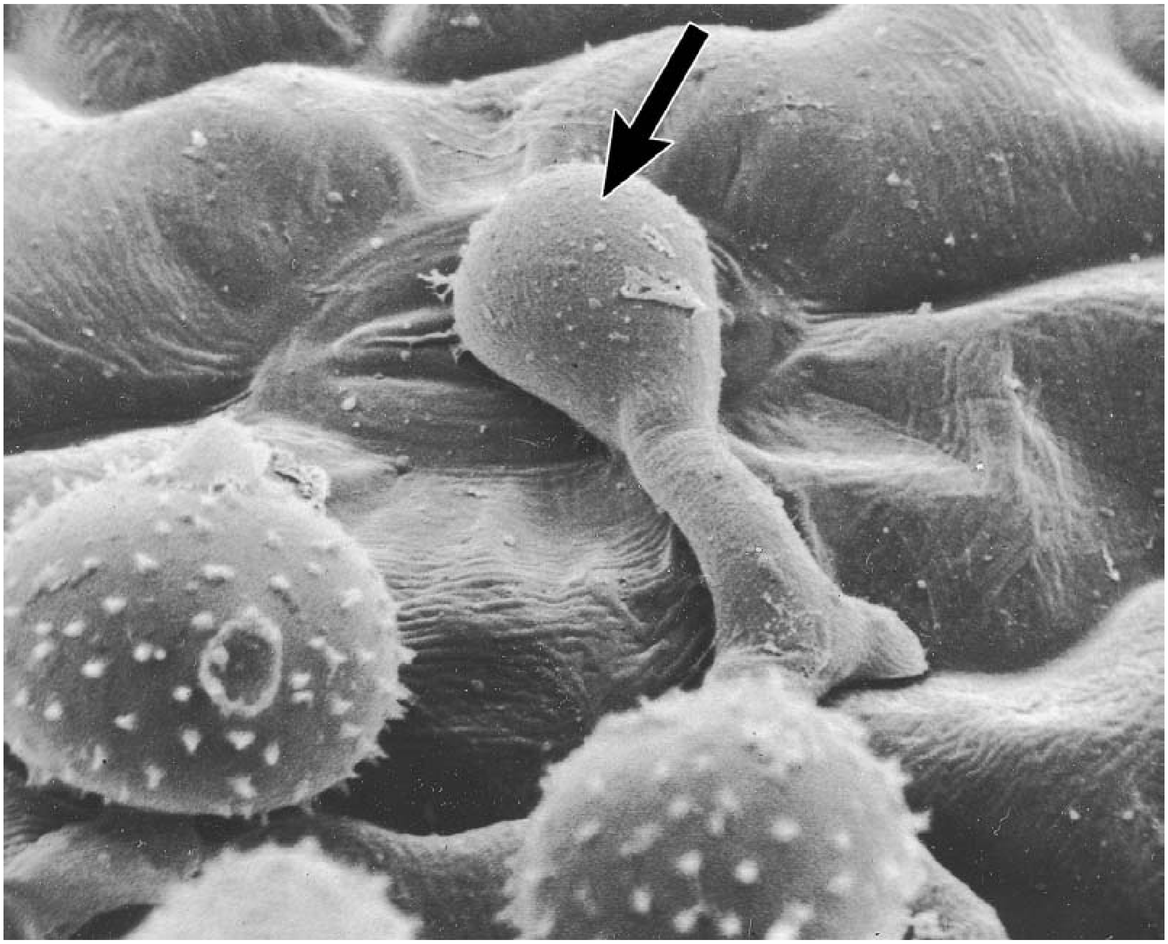


Figure 9.18.
The germ tube of a rust fungus spore grows to a stoma, produces an appressorium (arrow), and infects the leaf.

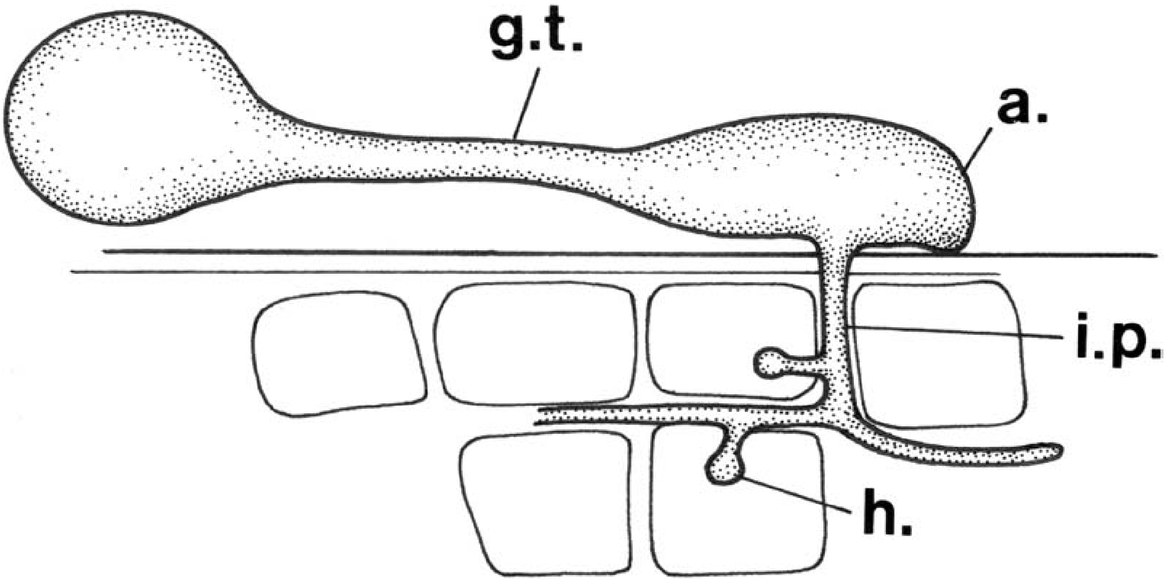


Figure 9.19.
Diagrammatic view of spore germination. a = appressorium, g.t. = germ tube, i.p. = infection peg, h = haustorium.

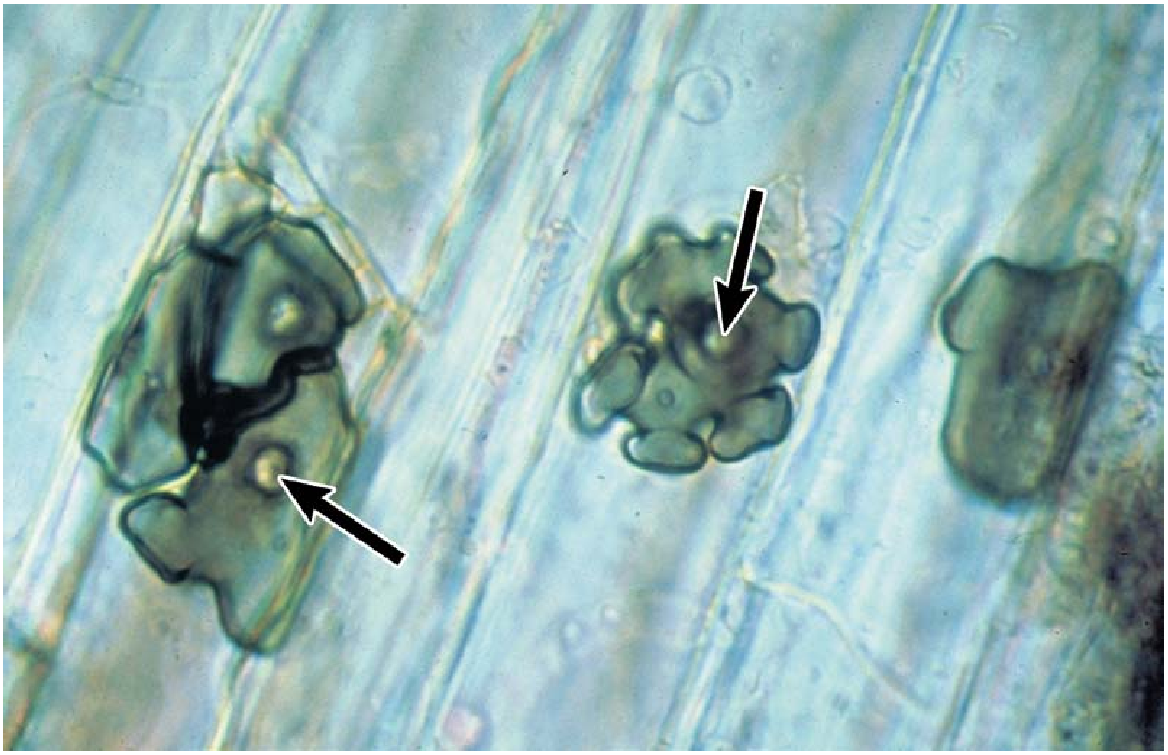


Figure 9.20.
Hyphopodia produced by a fungal pathogen (*Gaeumannomyces graminis* var. *graminis*) to infect roots. Note the infection pegs (arrows) in the centers of the lobes.

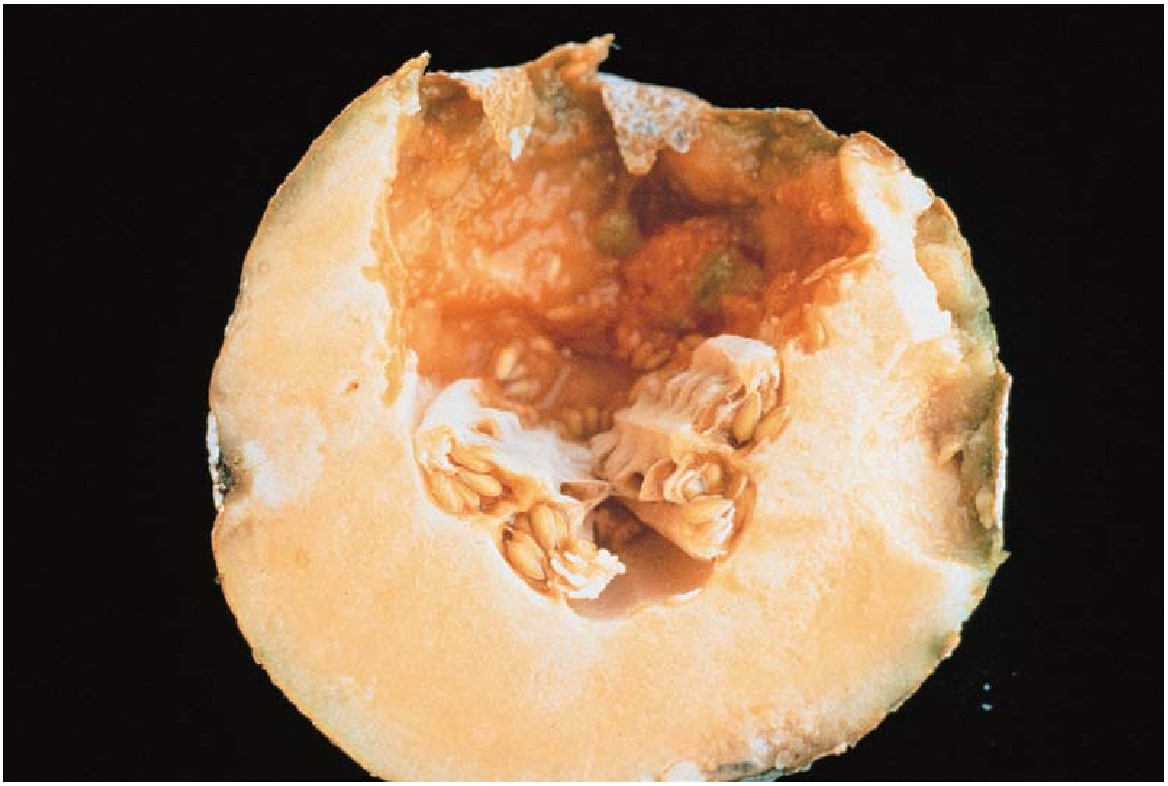


Figure 9.21.

Bacterial soft rot of melon, caused by *Erwinia carotovora* subsp. *carotovora* and the enzymes it produces.

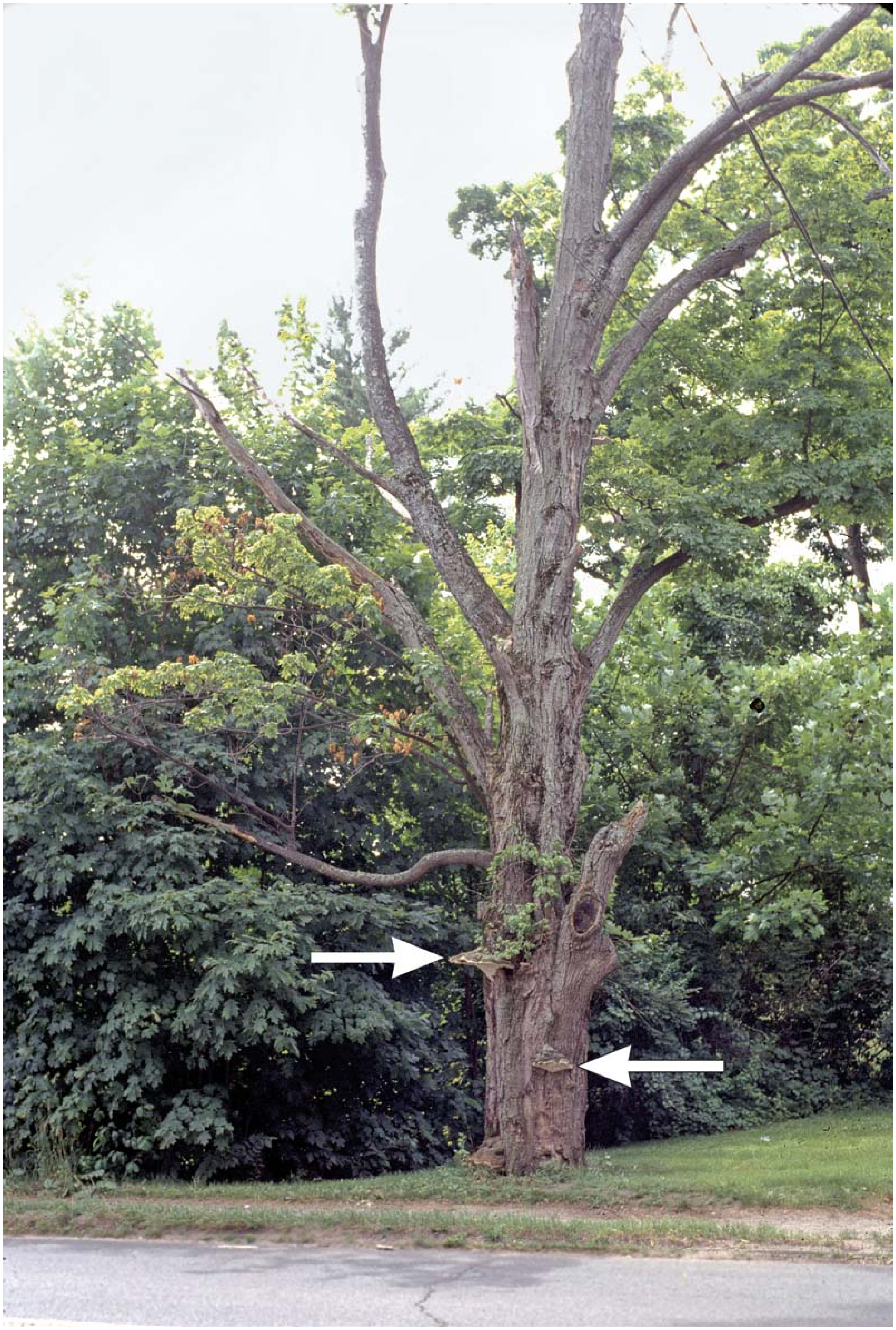


Figure 9.22.
Tree declining because of infection by a wood-decay fungus. Note the fruiting bodies (arrows) on the trunk of the tree. Wood-decay fungi produce lignases to degrade lignin.

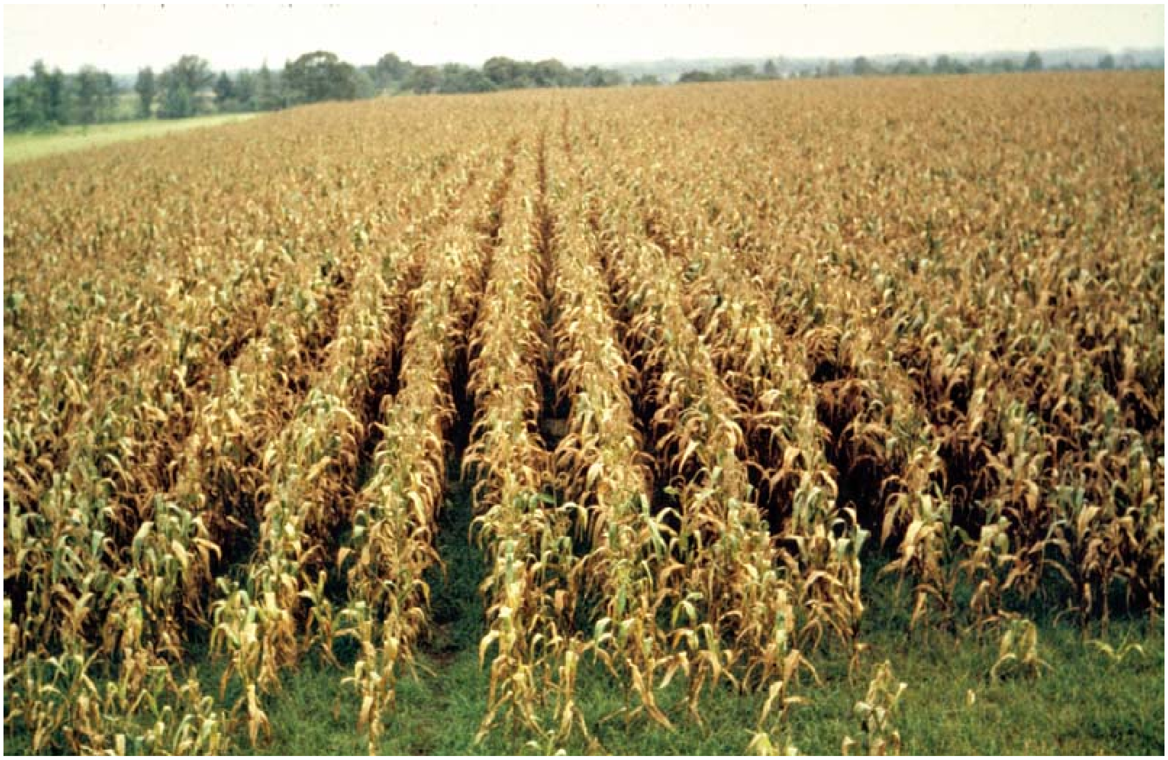


Figure 9.23.
Corn infected with race T of *Bipolaris maydis*, which produces a host-specific toxin. In the southern United States, the 1970 southern corn leaf blight epidemic destroyed the corn crop on many farms.

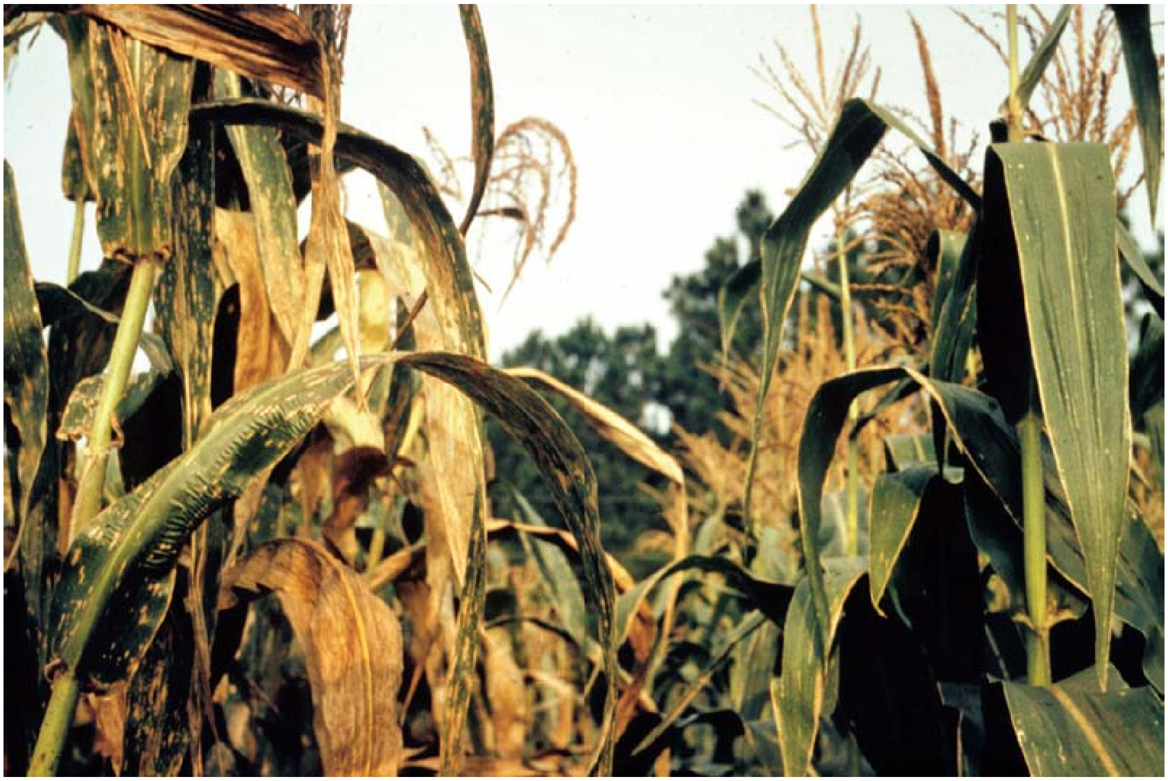


Figure 9.24.
A corn plant with Texas male-sterile cytoplasm (left) is very susceptible to the host-specific toxin produced by race T of the southern corn leaf blight (SCLB) pathogen. Corn with normal cytoplasm (right) is much less susceptible to SCLB.

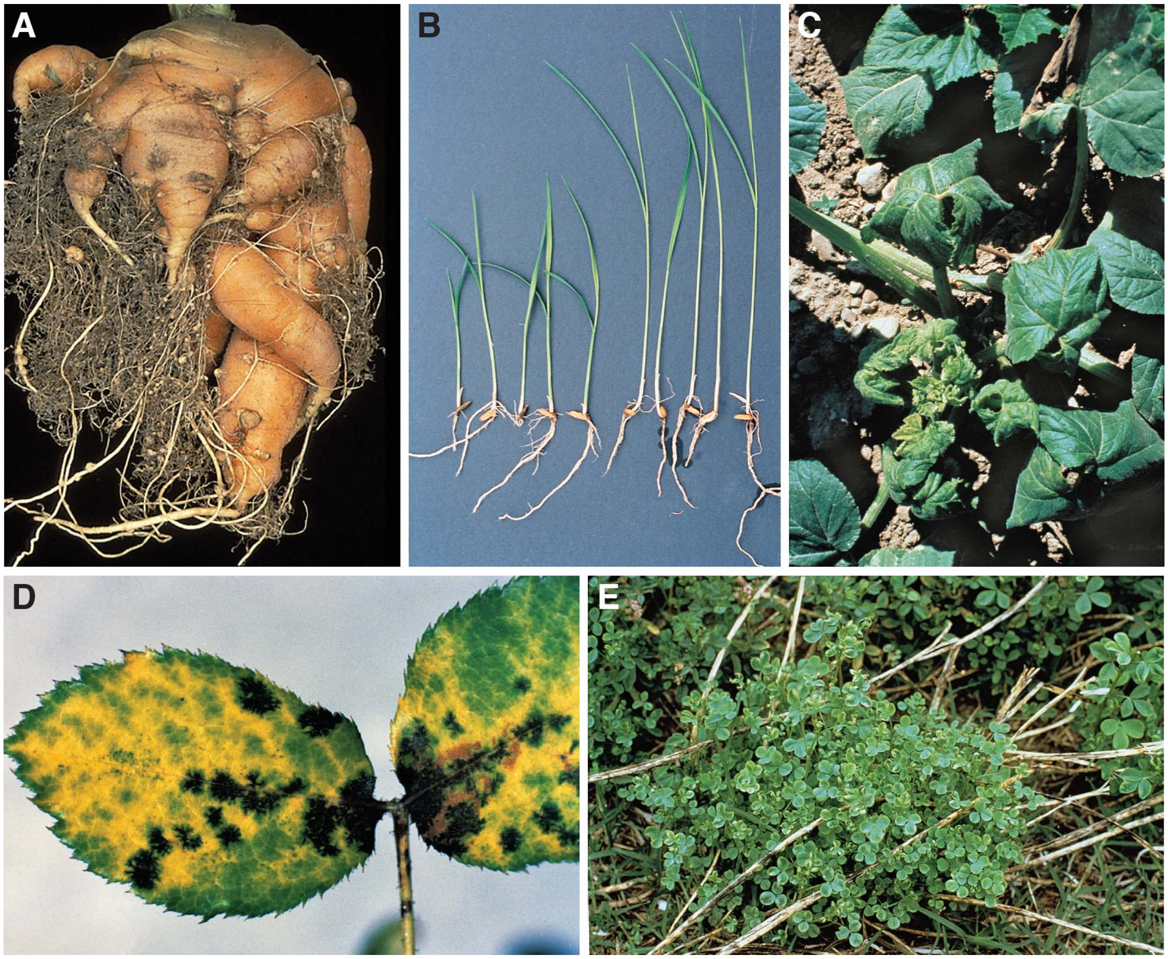


Figure 9.25.
Symptoms of plant diseases involving disruption of normal levels of growth regulators. **A**, Root galls and growth distortions of carrot caused by root-knot nematodes (*Meloidogyne* spp.). **B**, Rice with foolish seedling (bakanae) disease. Note etiolation of infected seedlings on the right due to gibberellin production by *Gibberella fujikuroi*. **C**, Summer squash infected by *Cucumber mosaic virus*. Note stunting and severe leaf distortion. **D**, Rose leaf with yellowing from ethylene production by a fungal pathogen, *Diplocarpon rosae* (black spot disease). **E**, Witches' broom of alfalfa caused by a phytoplasma. Note the proliferation of stems and the pale foliage.

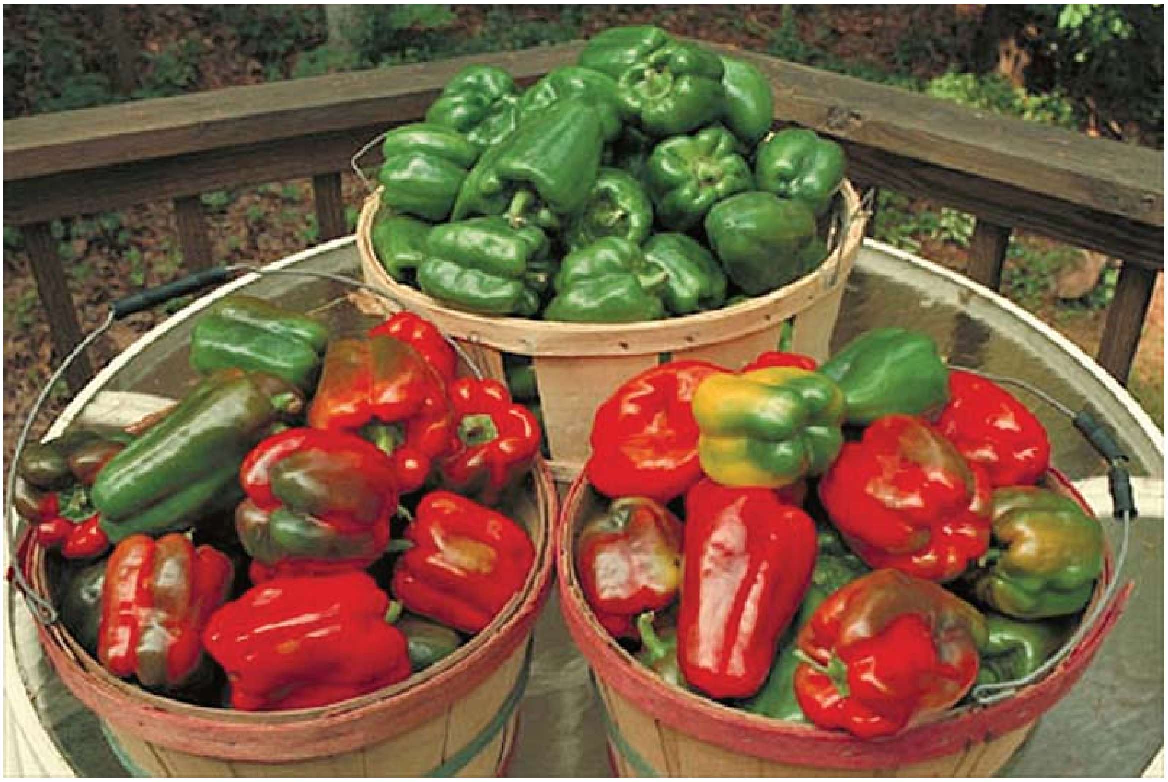


Figure 9.26.
The waxy surface of these bell peppers helps reduce surface water and infection by fungi and bacteria.

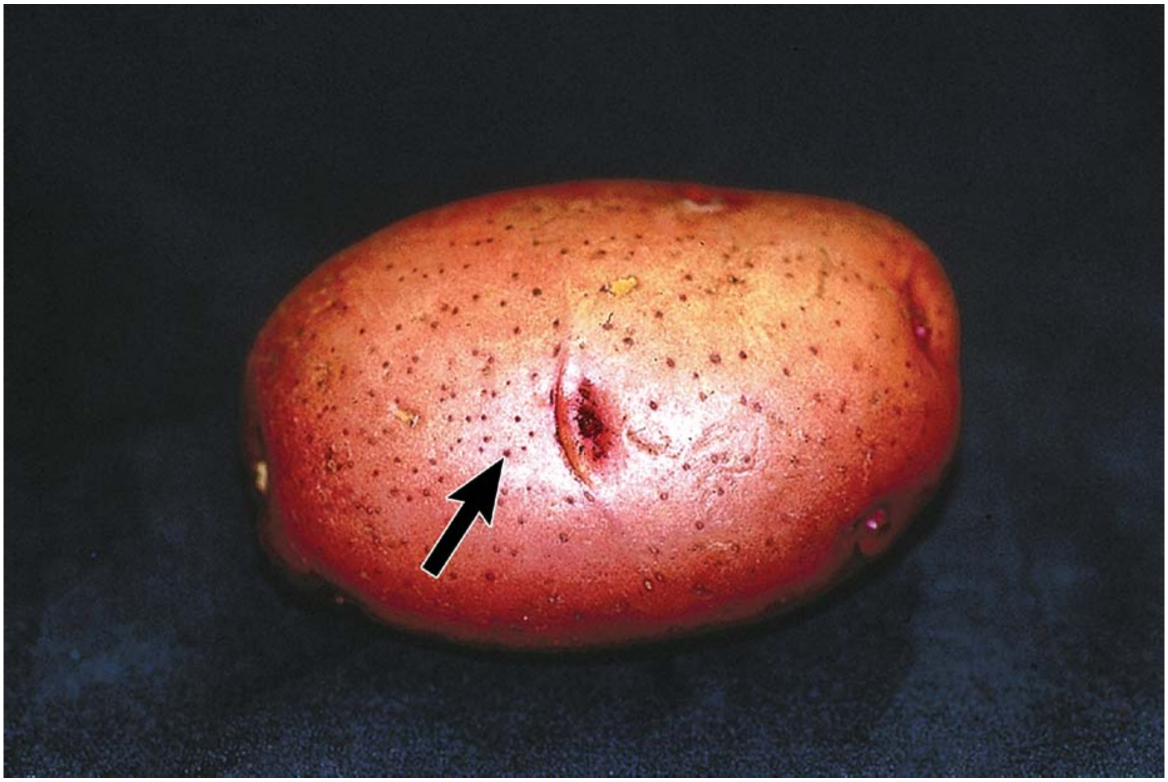


Figure 9.27.
Most surface cells of potato tubers are suberized and provide a good defense against many pathogens. Potato tuber lenticels (arrow) are usually inconspicuous, but they become enlarged in wet soil, allowing entry by pathogens.

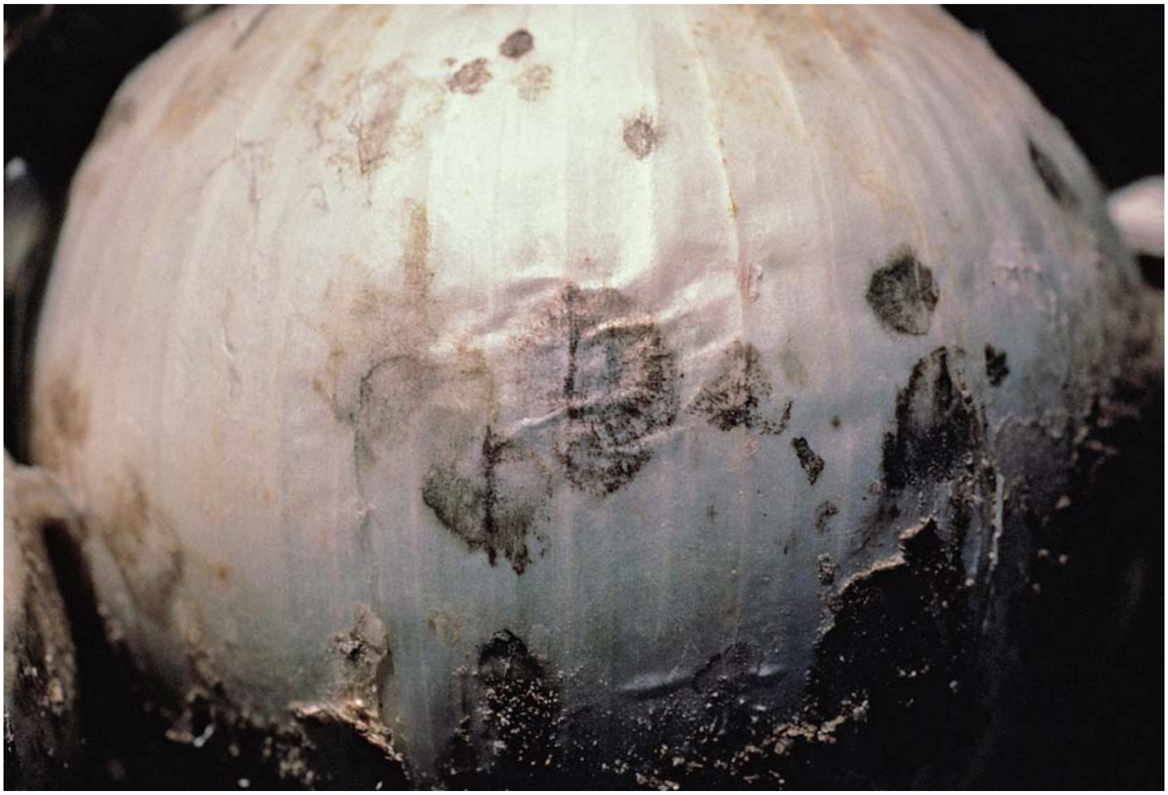


Figure 9.28.
Onion smudge, a fungal disease, on a white onion. Red onions are more resistant to smudge because of the chemical inhibitors in the colored bulb scales.

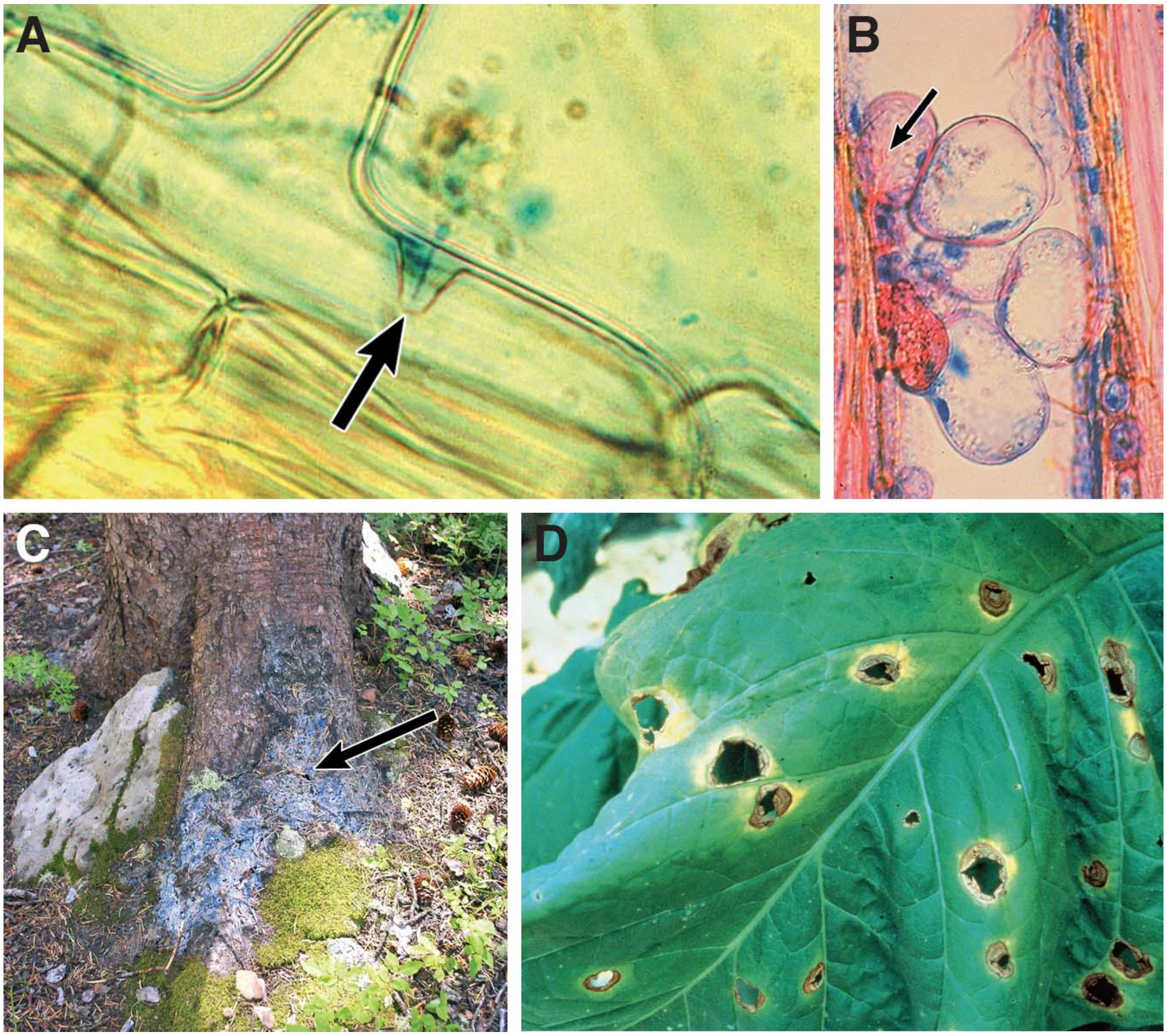
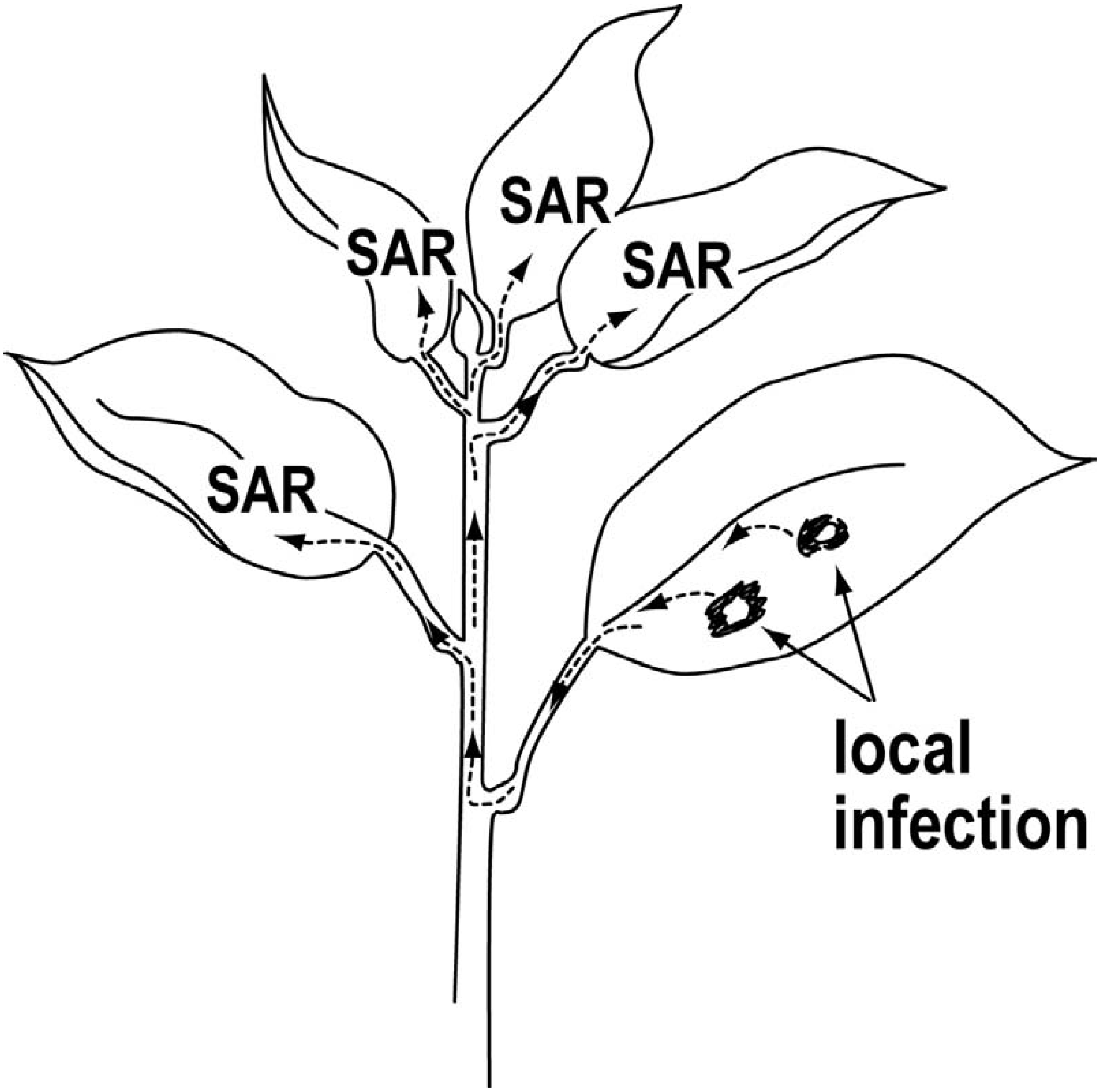


Figure 9.29.

Some examples of induced structural defenses. **A**, Papilla (arrow) formed around a fungal hypha at the site of attempted infection. **B**, Longitudinal section of an elm vessel with tyloses (arrow) that formed in response to Dutch elm disease. **C**, Resin (arrow) production in conifer with Armillaria root disease. **D**, Shot-hole symptom in tobacco, caused by the fungus *Cercospora nicotianae*. The formation of a circular abscission layer causes the infected part to fall out.



SAR
SAR
SAR
SAR
local infection

Figure 9.30.
Systemic acquired resistance (SAR) is induced in the new growth of a plant that has been exposed to a pathogen.

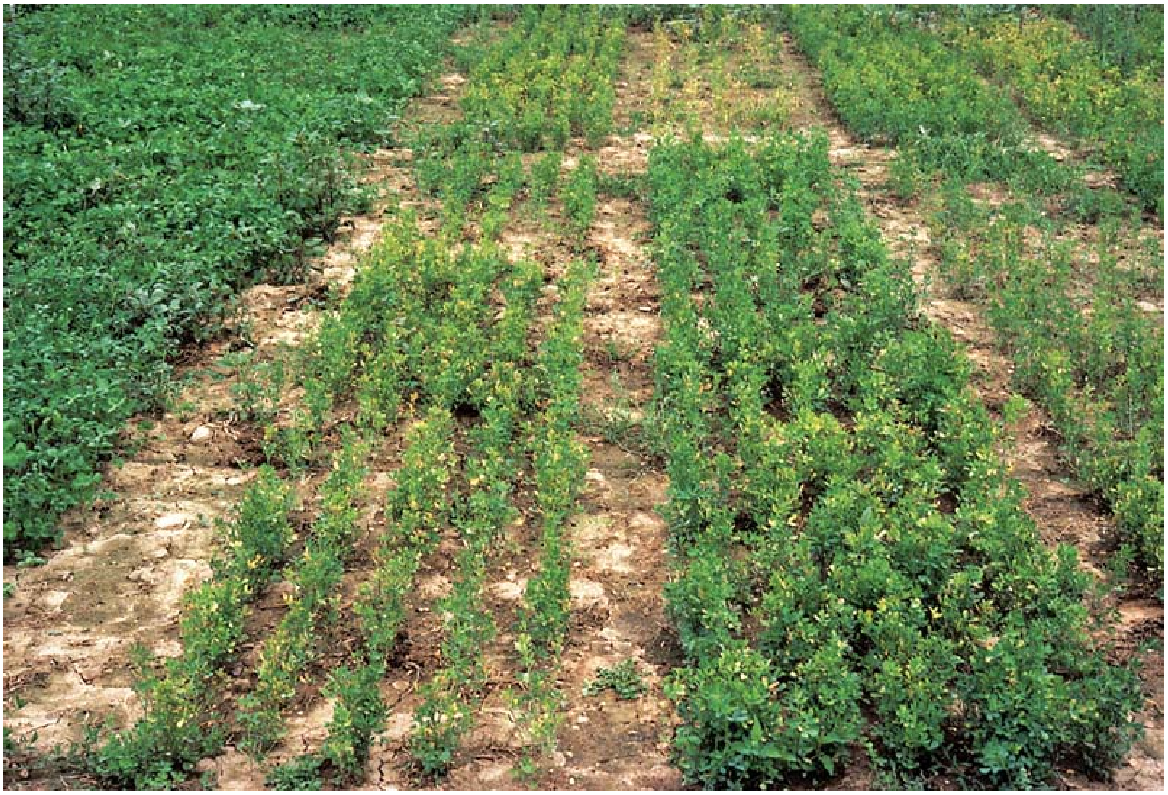


Figure 9.31.
Alfalfa lines susceptible (left) and resistant (right) to Aphanomyces root rot in naturally infested soil.

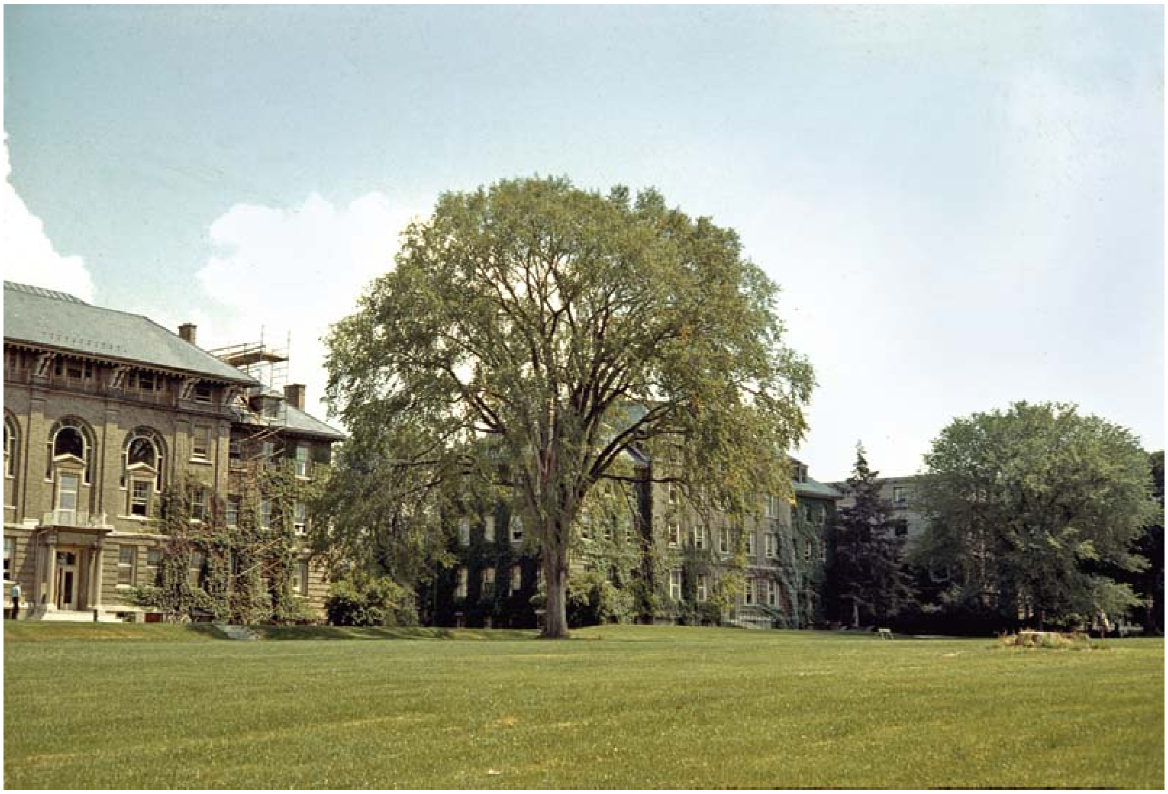


Figure 9.32.
An elm tree that has escaped Dutch elm disease despite the death and removal of many nearby elms.

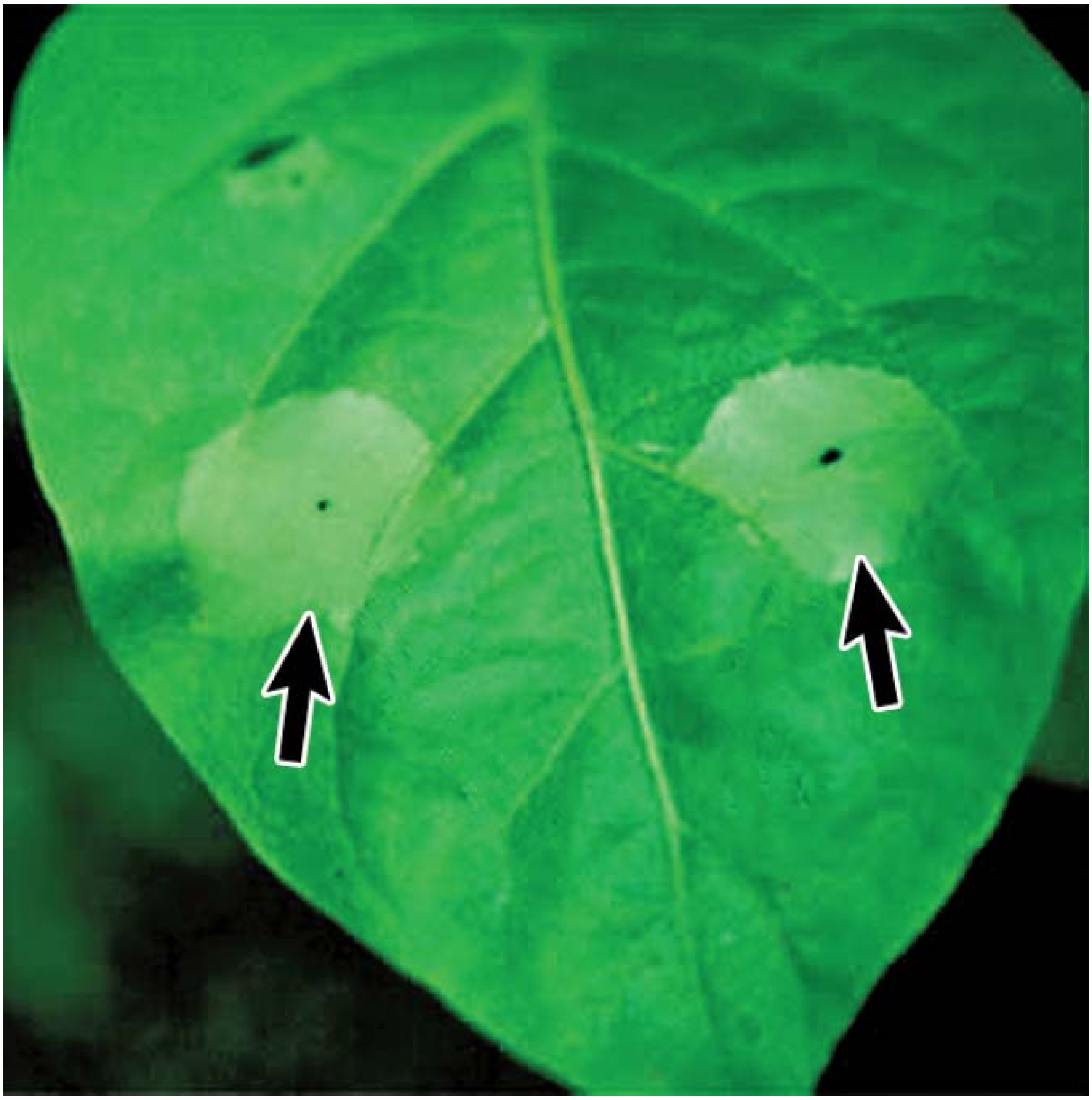


Figure 9.33.
Areas (arrows) of hypersensitive response (HR) in pepper in response to infection by a bacterial spot pathogen.

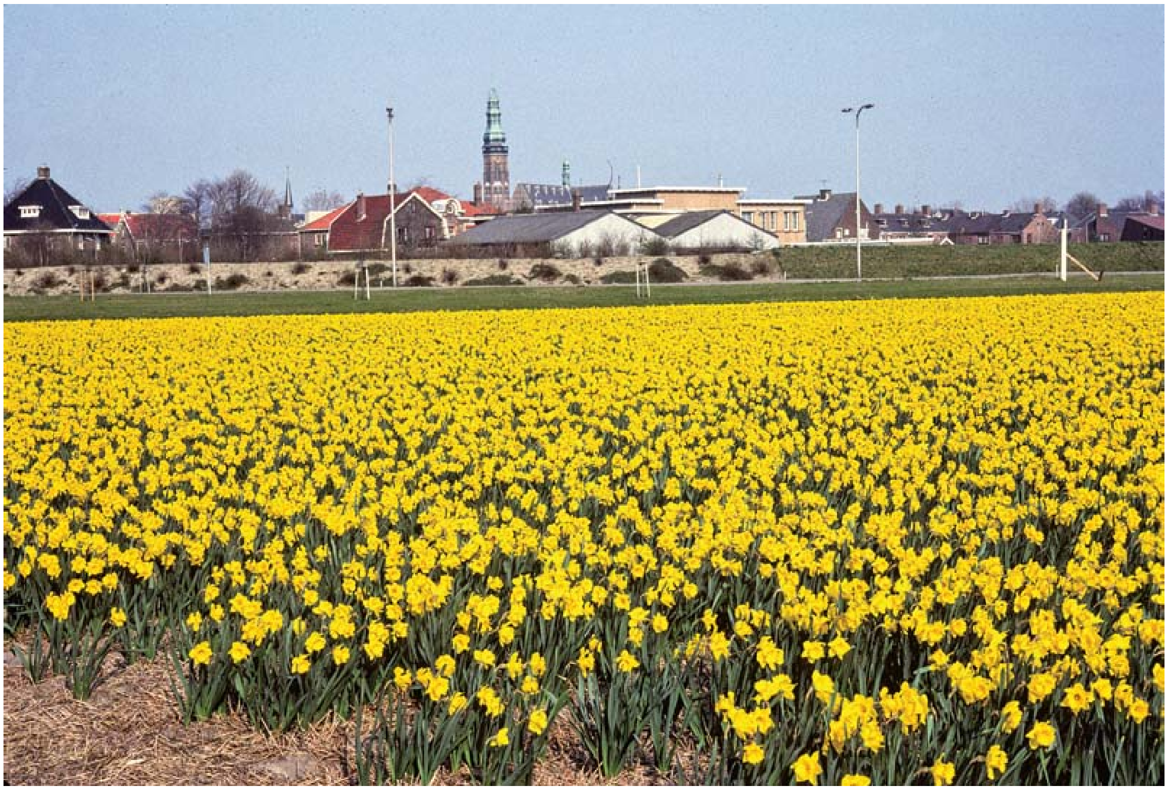


Figure 9.34.
Field of genetically uniform daffodils. Gardeners prefer uniform flowers, but these plants are all equally susceptible to potential pathogens.

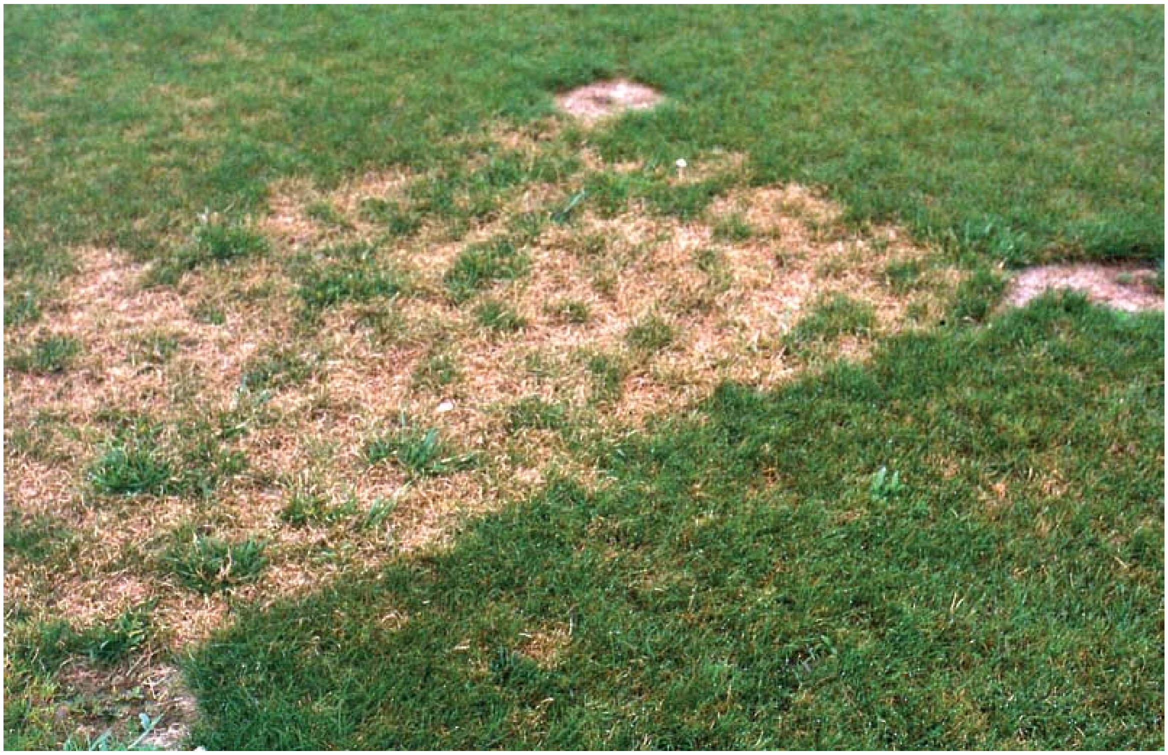


Figure 9.35.
Variation in resistance to dollar spot among cultivars of fine fescues. The demand for uniformity in turfgrass growth characteristics also results in uniform disease resistance or susceptibility.

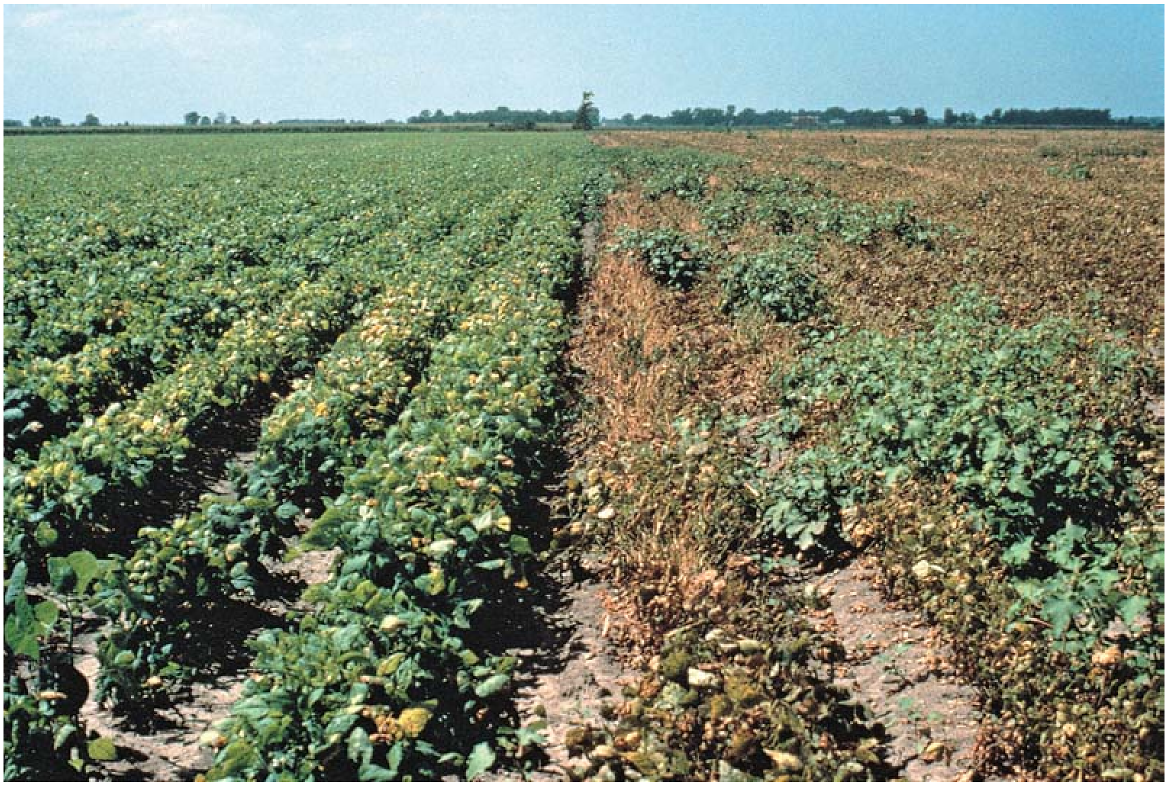


Figure 9.36.
Beans resistant (left) and susceptible (right) to the prevalent race of rust caused by *Uromyces appendiculatus*.

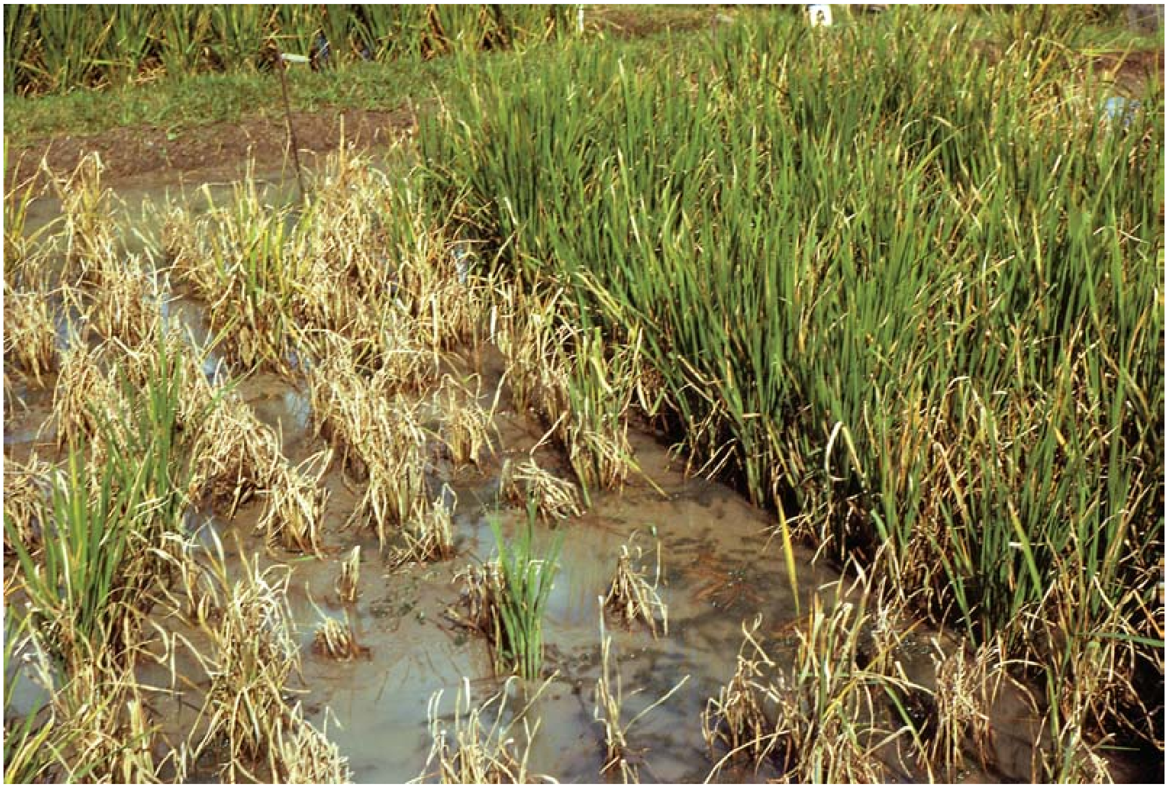


Figure 9.37.
Rice susceptible (left) and resistant (right) to tungro virus disease.

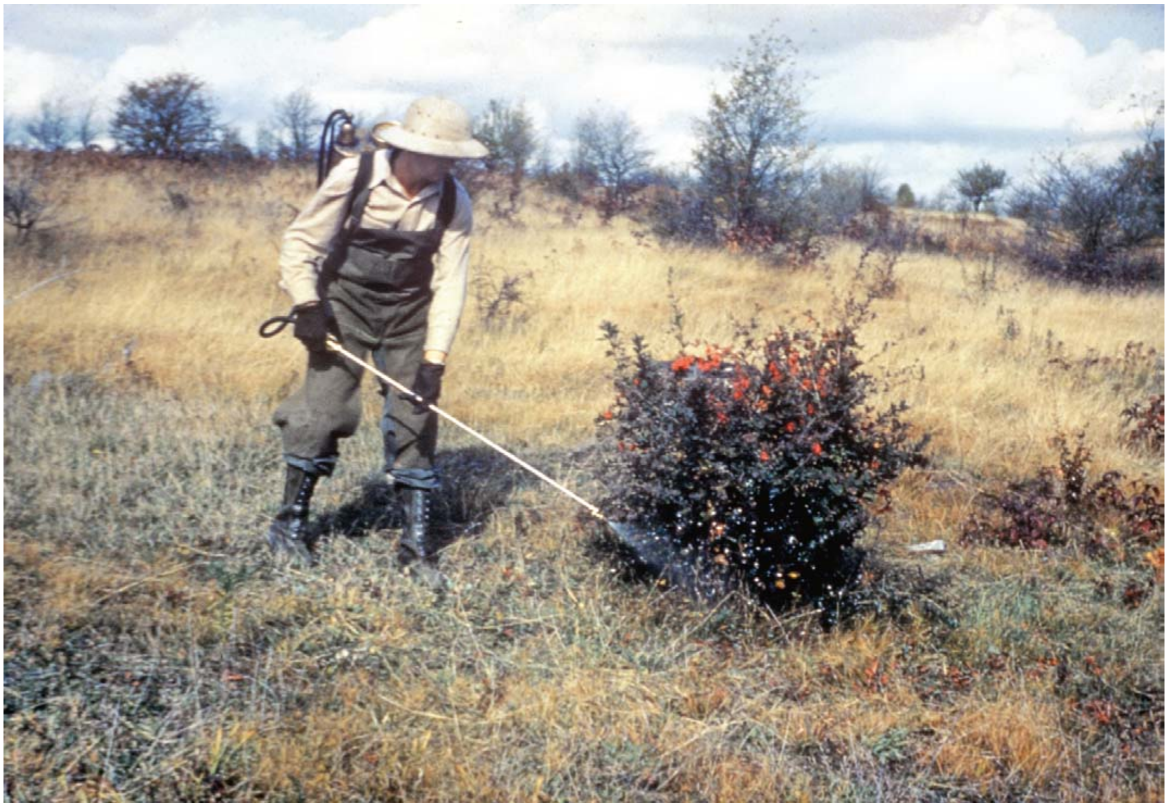


Figure 9.38.
Spraying common barberry with herbicide to eliminate the alternate host of *Puccinia graminis*. This reduces the genetic variation in this important rust fungus by eliminating the sexual cycle.

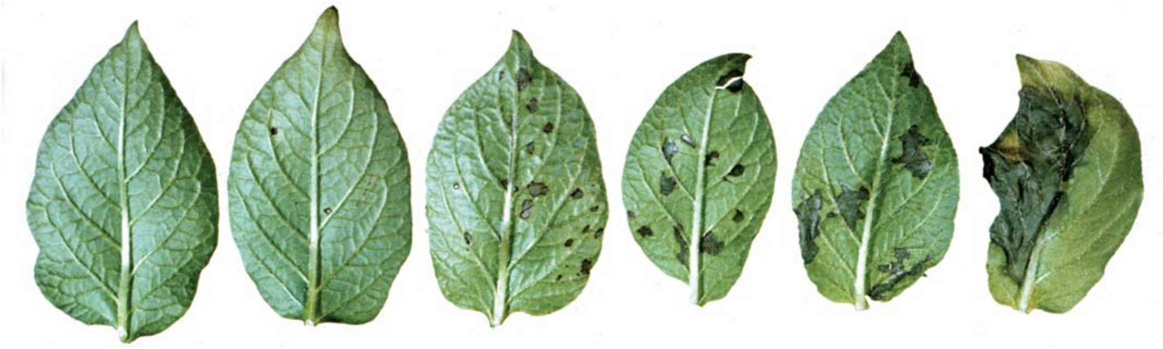


Figure 9.39.
Potato leaf from a susceptible plant (right) and leaves from plants with varying levels of general resistance. General resistance functions against all races of the late blight pathogen, *Phytophthora infestans*. In resistant plants, lesions are smaller, develop more slowly, and produce fewer sporangia.

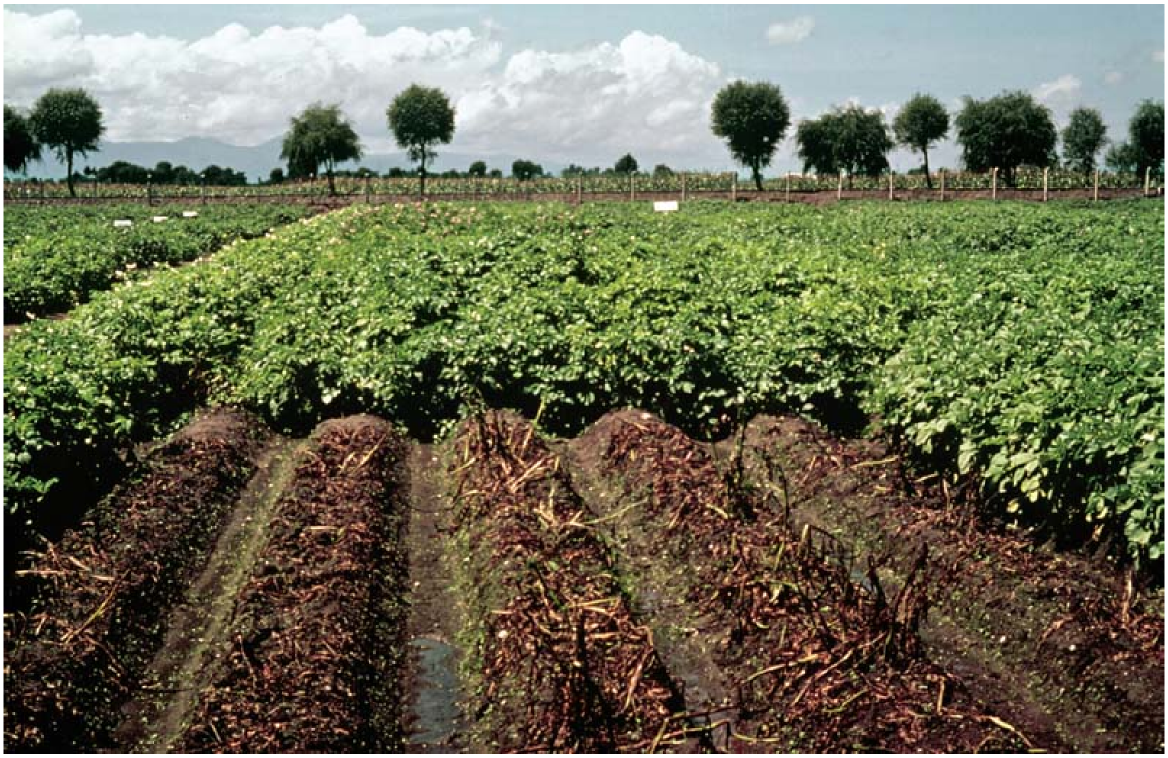


Figure 9.40.
Potatoes with general resistance to late blight. Note dead plants of a commercial cultivar in the foreground.

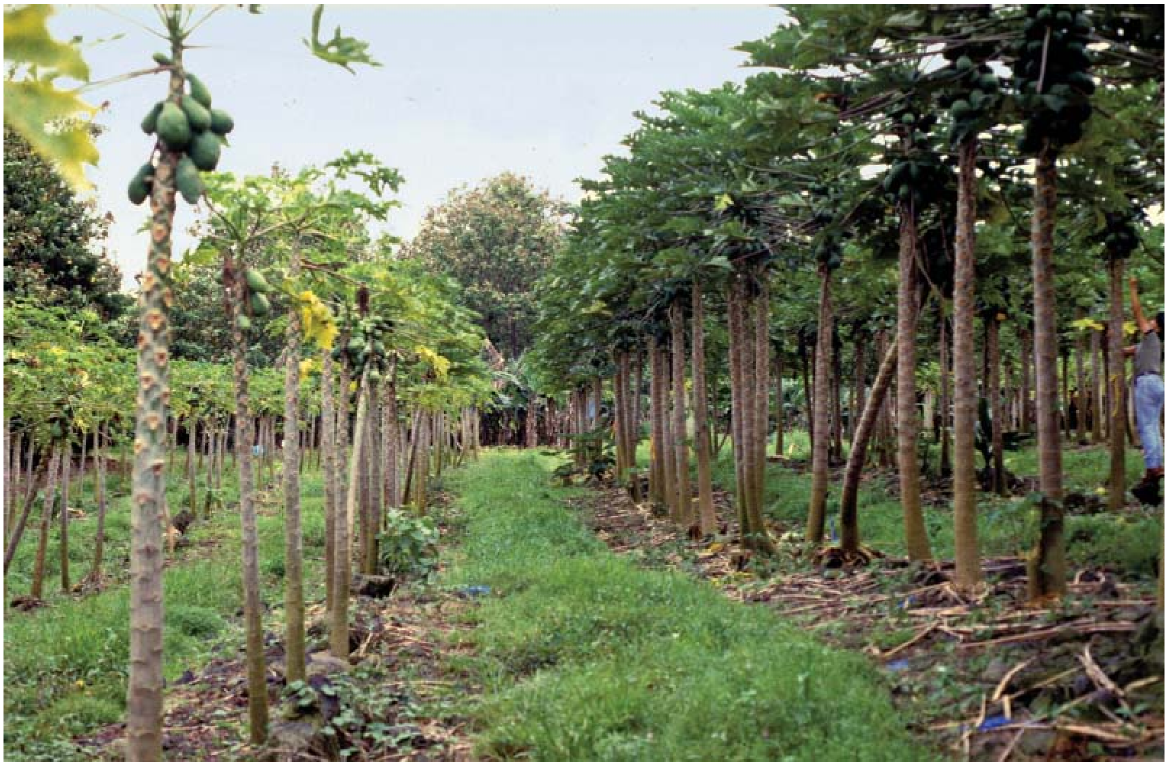


Figure 9.41.
Transgenic papaya resistant to *Papaya ringspot virus* (PRSV) (right) and susceptible papaya (left). Plant embryos received the coat protein gene of PRSV by biolistic transformation.

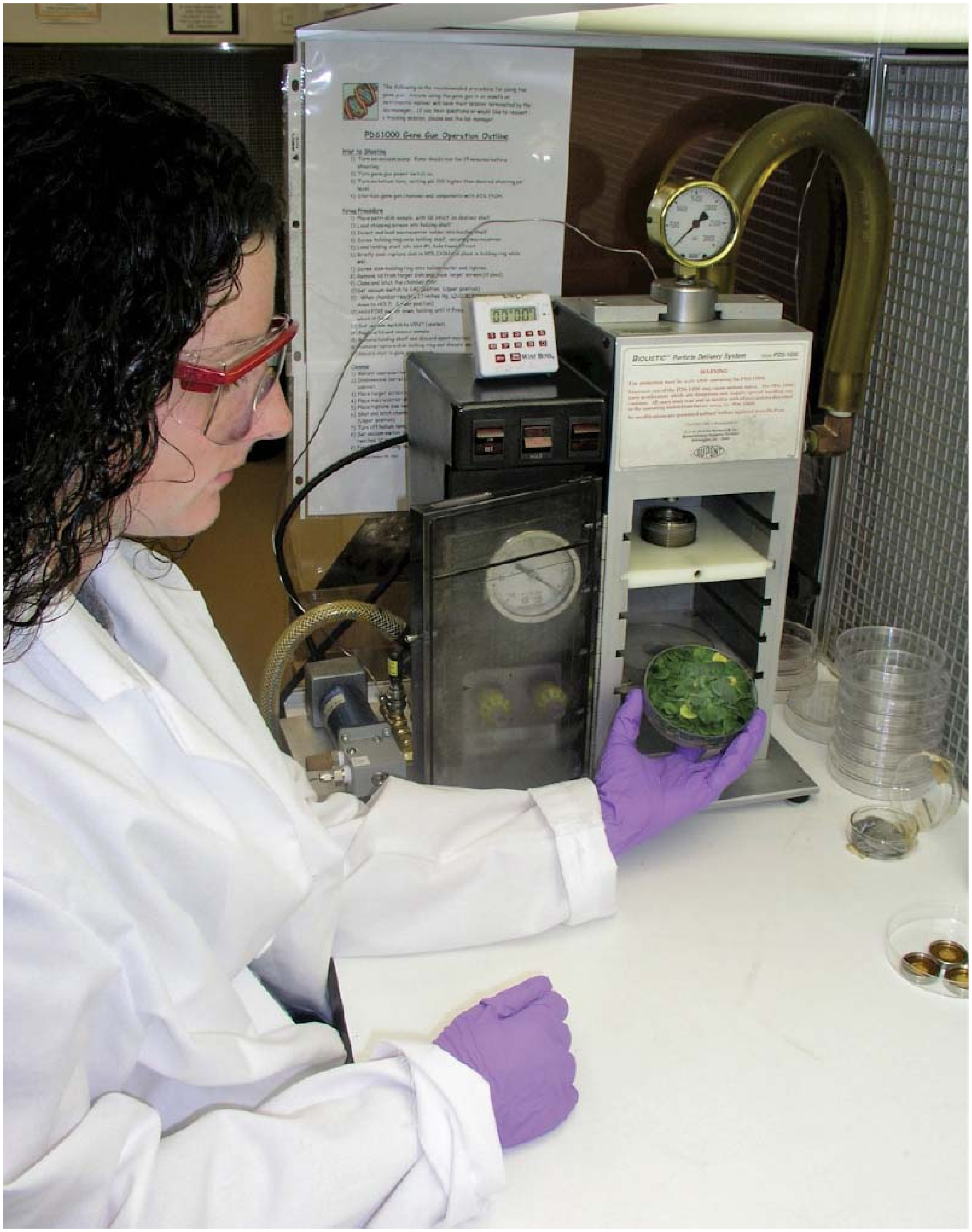


Figure 9.42.
The “gene gun” used for biolistic transformation of plants.

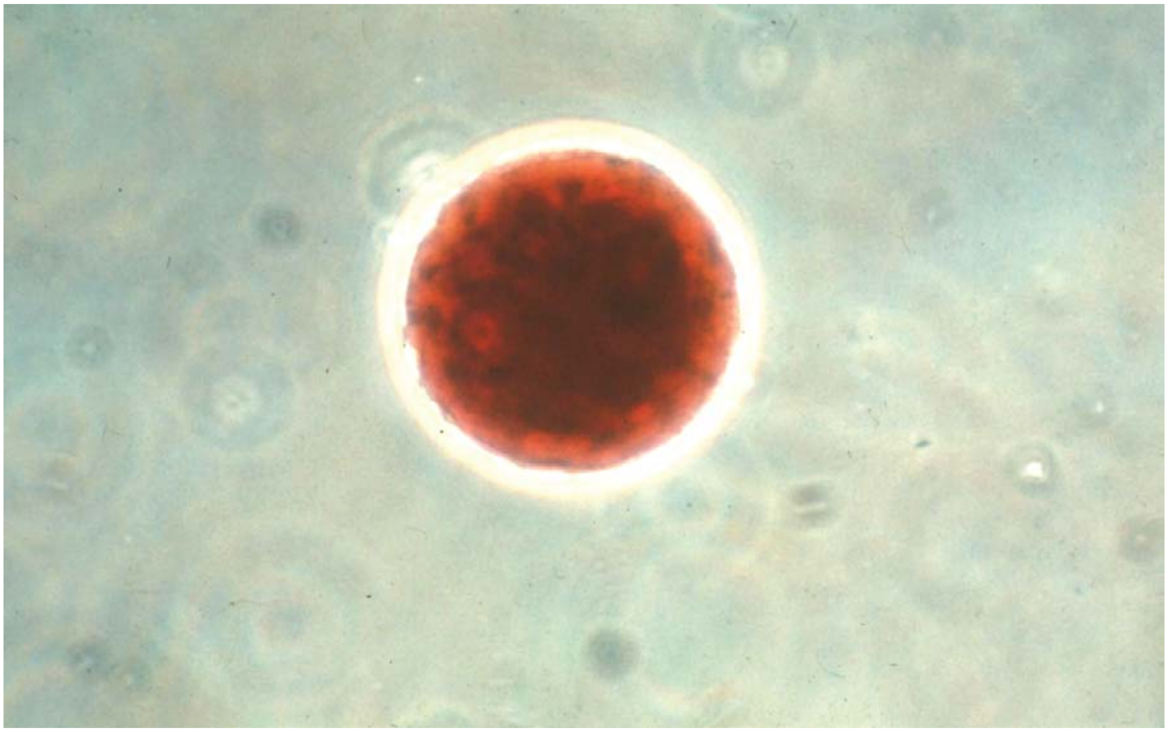
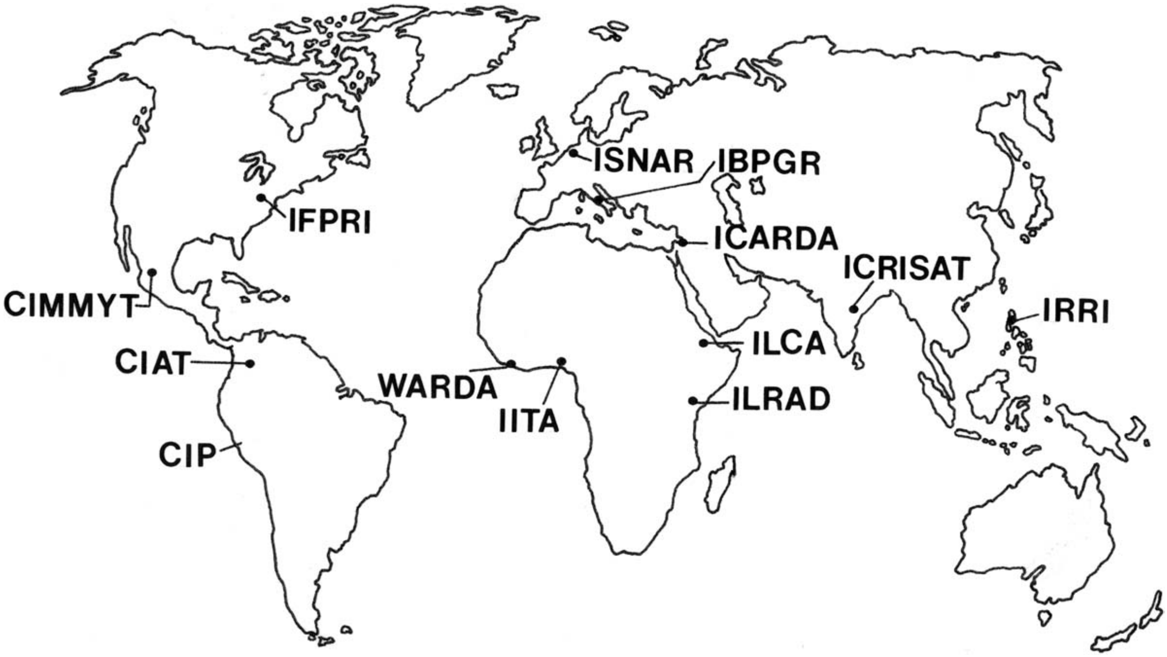


Figure 9.43.
Protoplast of a plant cell from which the cell wall has been removed enzymatically, leaving only the cell membrane to enclose the cell contents.


ISNAR
IBPGR
IFPRI
ICARDA
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CIAT
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WARDA
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IITA
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Figure 9.44.
The international agricultural research centers.

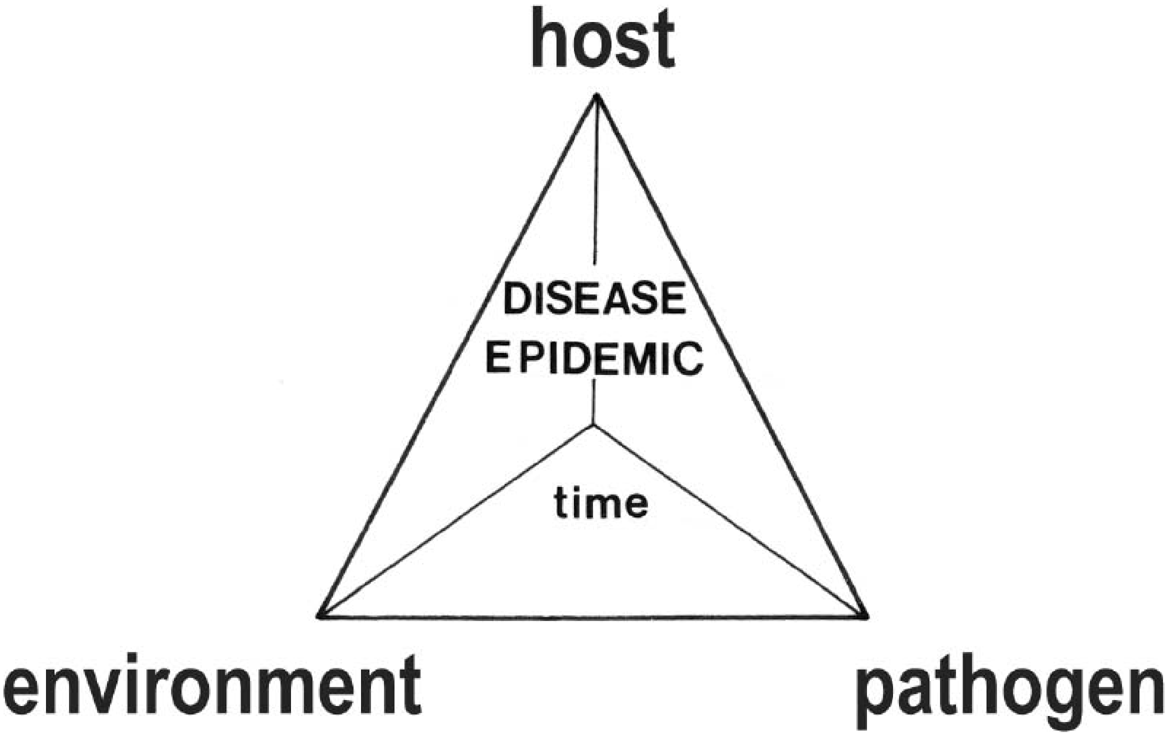

host
DISEASE
EPIDEMIC
time
environment
pathogen

Figure 10.1.
The plant disease pyramid.

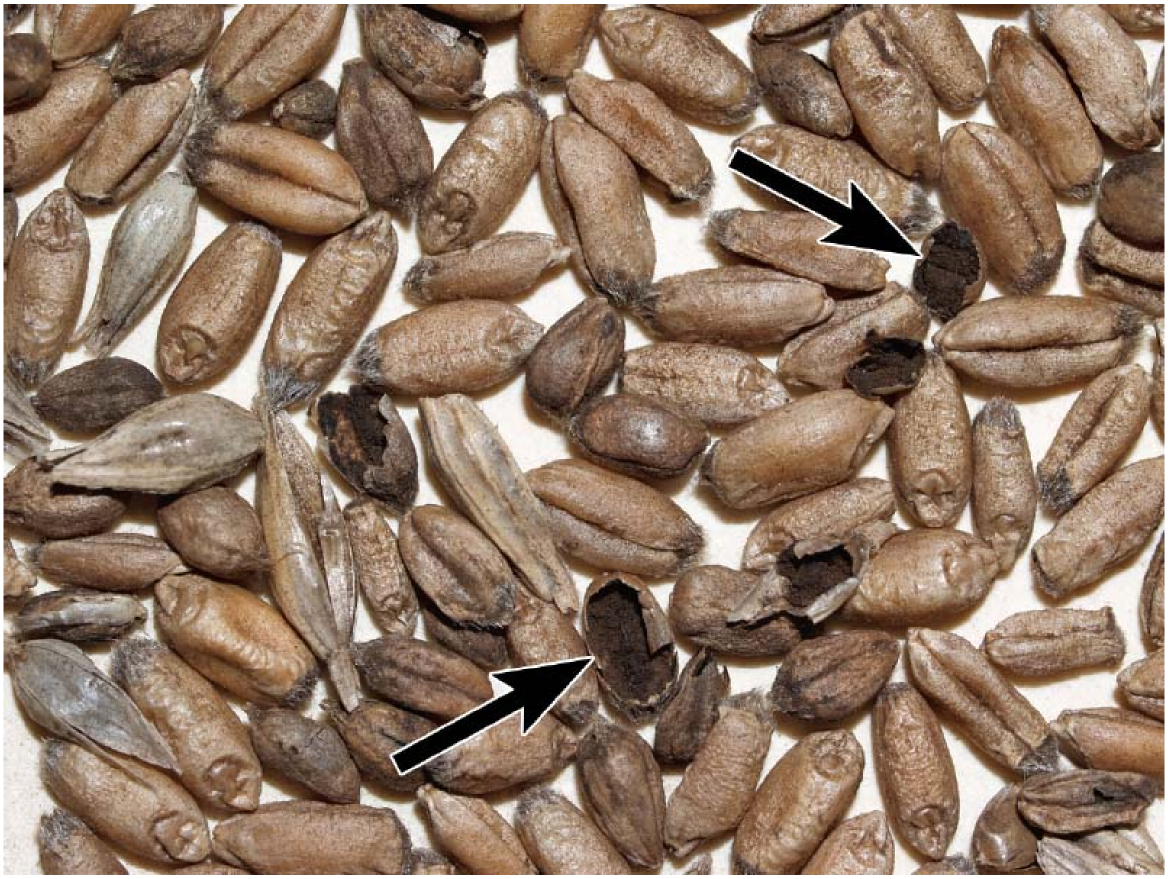


Figure 10.2.
Masses of teliospores (arrows) from bunted wheat kernels with stinking smut, a monocyclic disease.

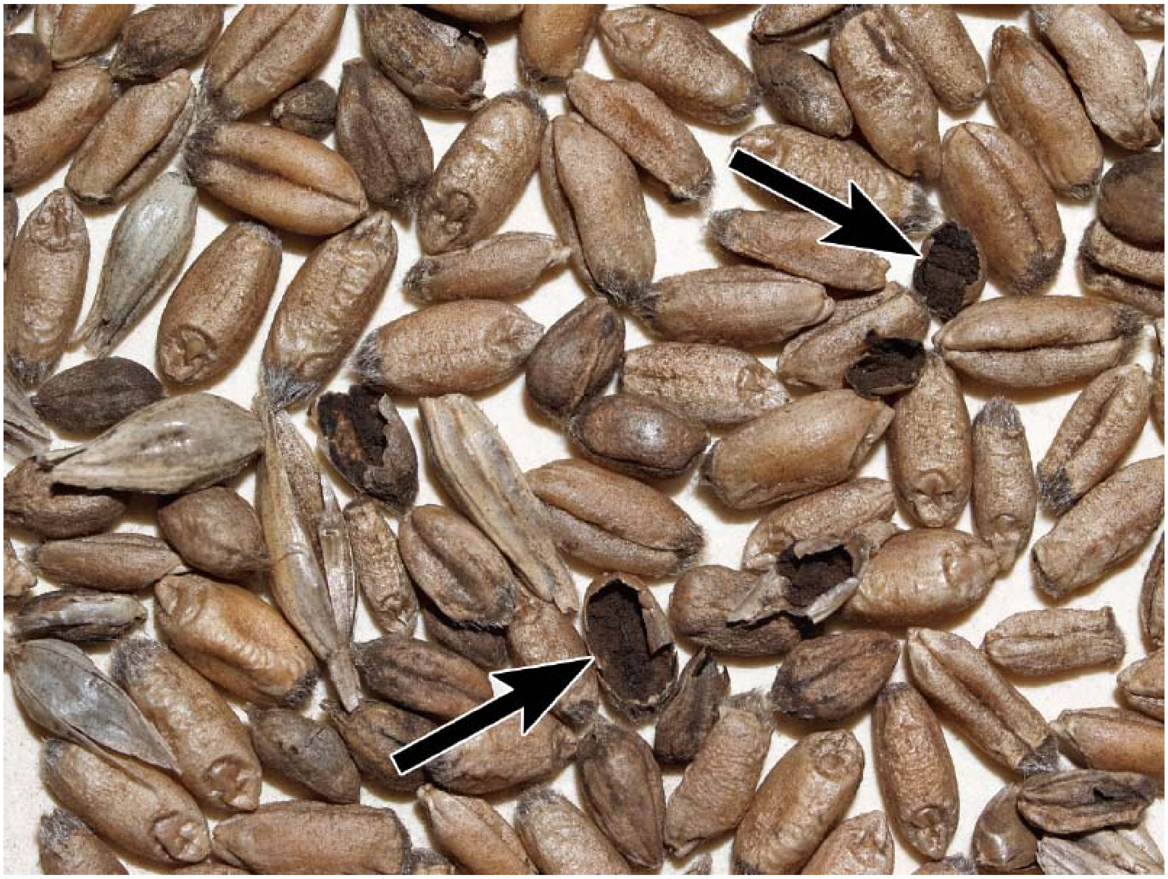


Figure 10.3.
Coffea arabica (left) defoliated by coffee rust, a polycyclic disease, compared with a resistant hybrid of *C. arabica* and *C. canephora* (right).

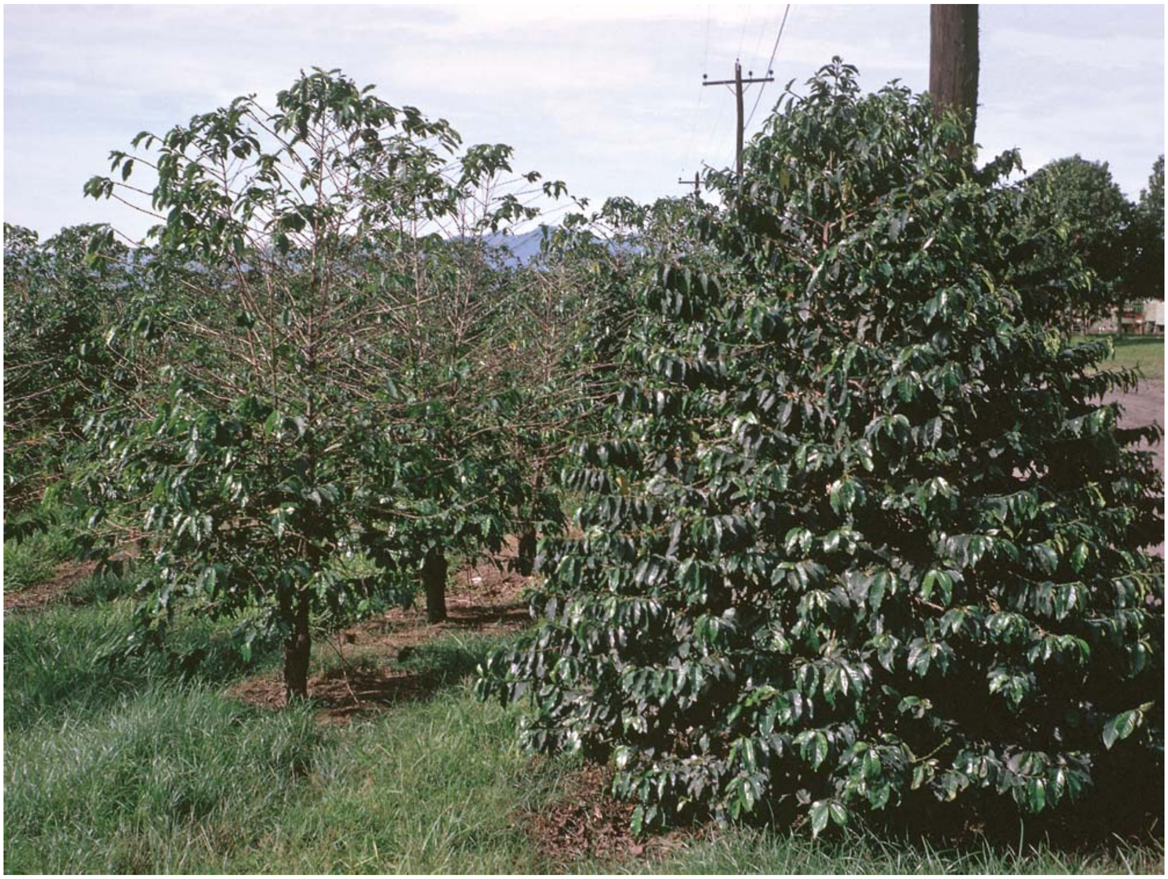


Figure 10.4.
Early blight (*Alternaria solani*) of potato and tomato, a polycyclic, foliar fungal disease. Note the concentric rings in the lesions, characteristic of *Alternaria* diseases.

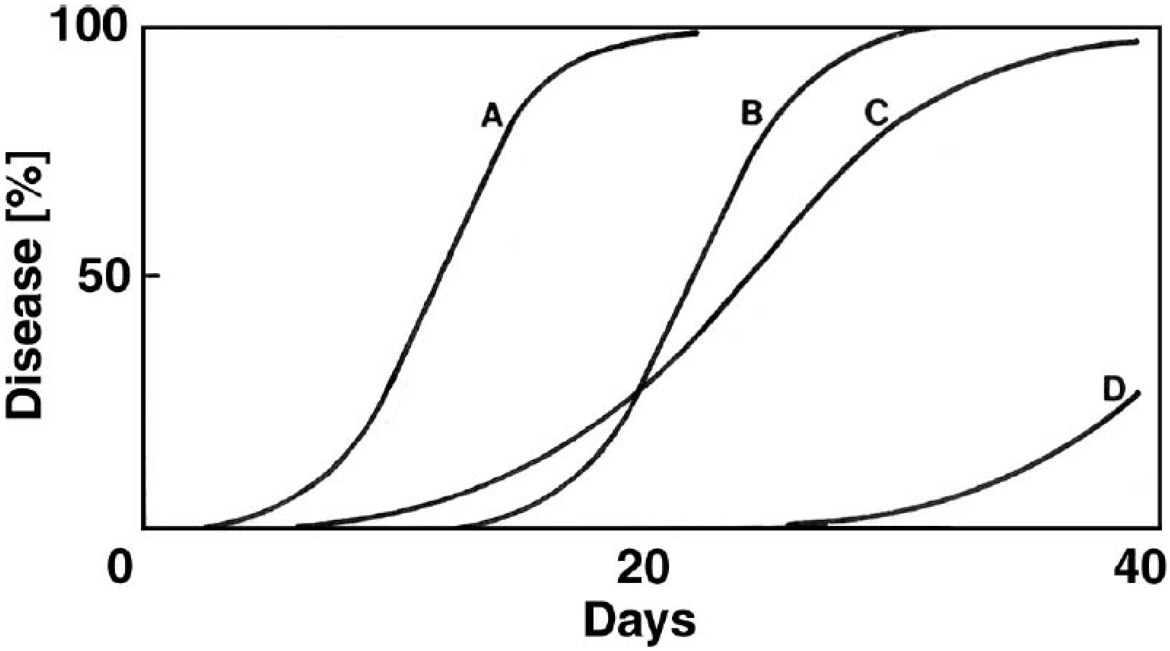


Figure 10.5.
Disease progress curves of a polycyclic pathogen under various conditions. A = no management. B = reduction of primary inoculum, which delays the epidemic but does not reduce the rate of infection. C = reduction in the rate of infection. D = combination of B and C, i.e., the epidemic is delayed and the rate of infection is reduced.

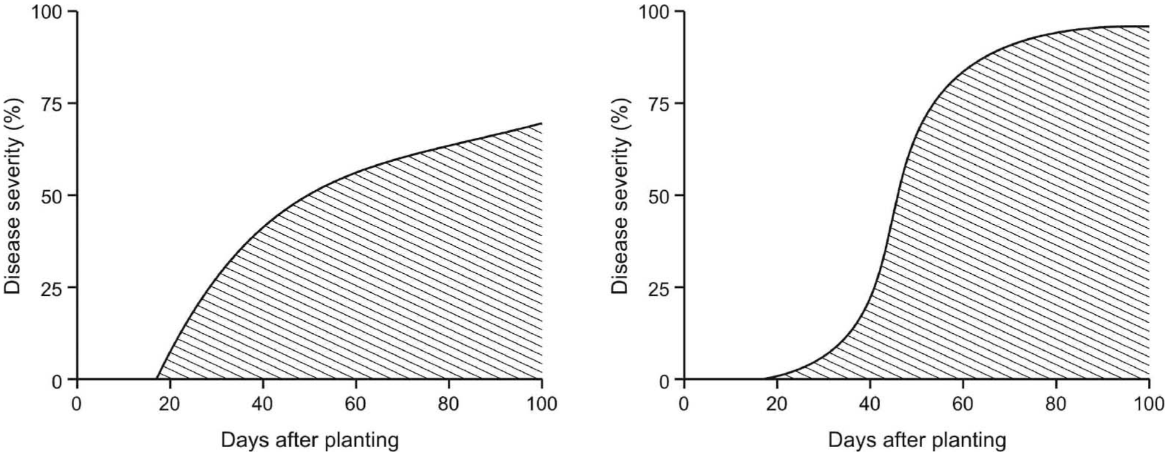


Figure 10.6.

Epidemics can be compared by calculating the area under the disease progress curve (AUDPC, shaded area) for each epidemic. These are two examples.

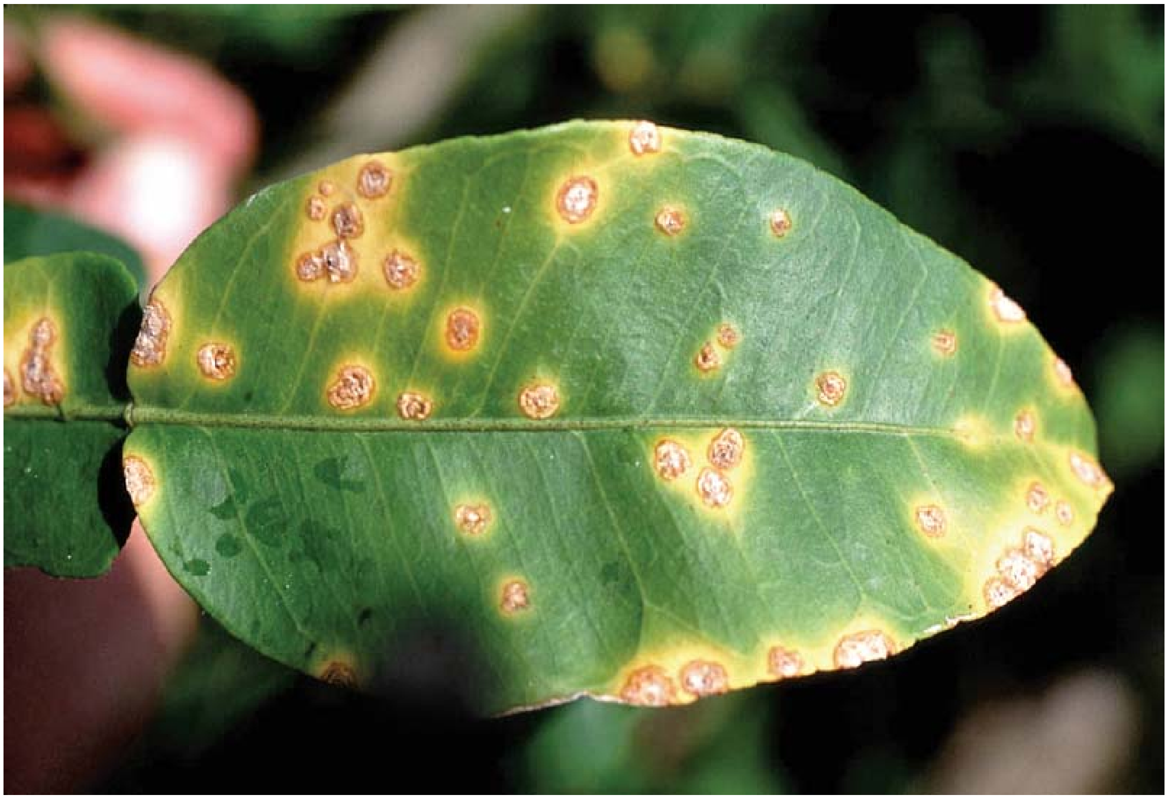


Figure 10.7.
Foliar symptoms of citrus canker, a polycyclic disease. Note the halo around each lesion, typical of a bacterial disease.

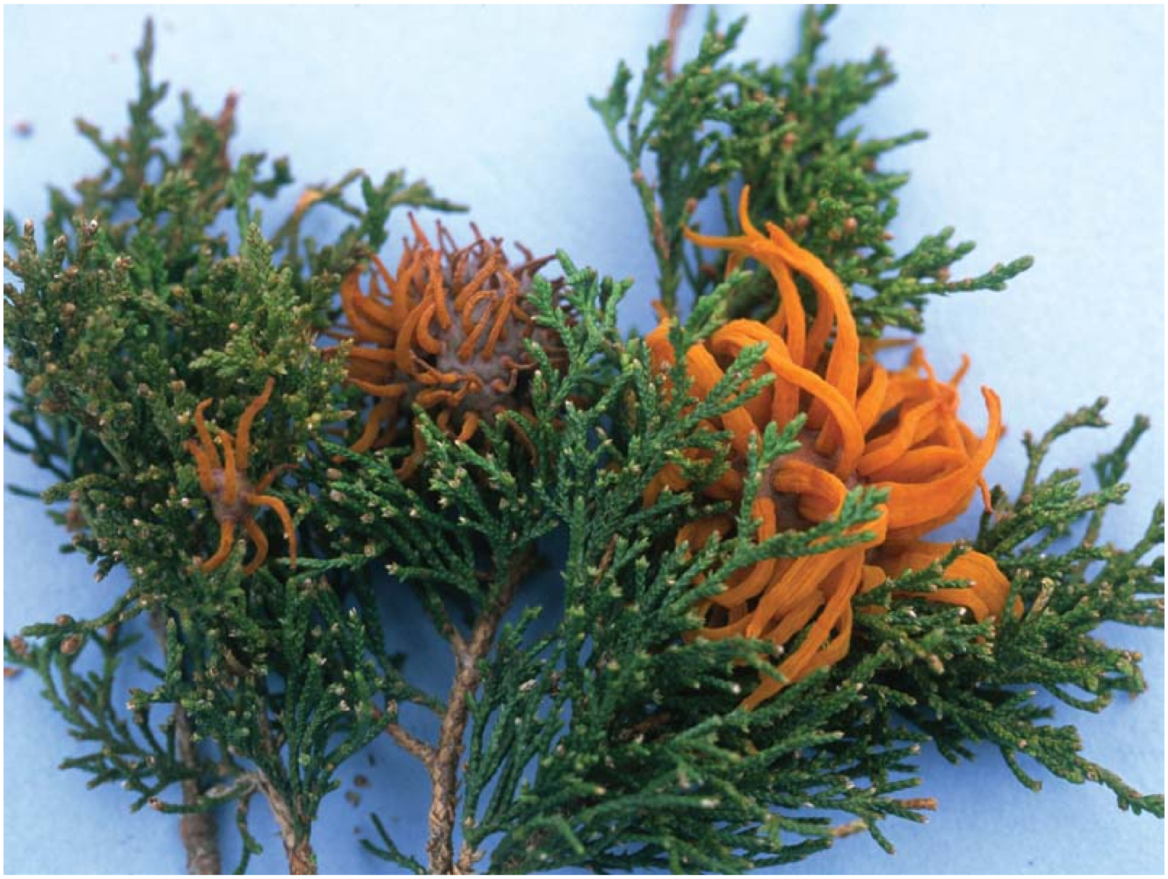


Figure 10.8.
The production of basidiospores from telial galls of the cedar-apple rust fungus on juniper (red-cedar) is used to determine the need for and the timing of application of protective fungicides to apple trees.

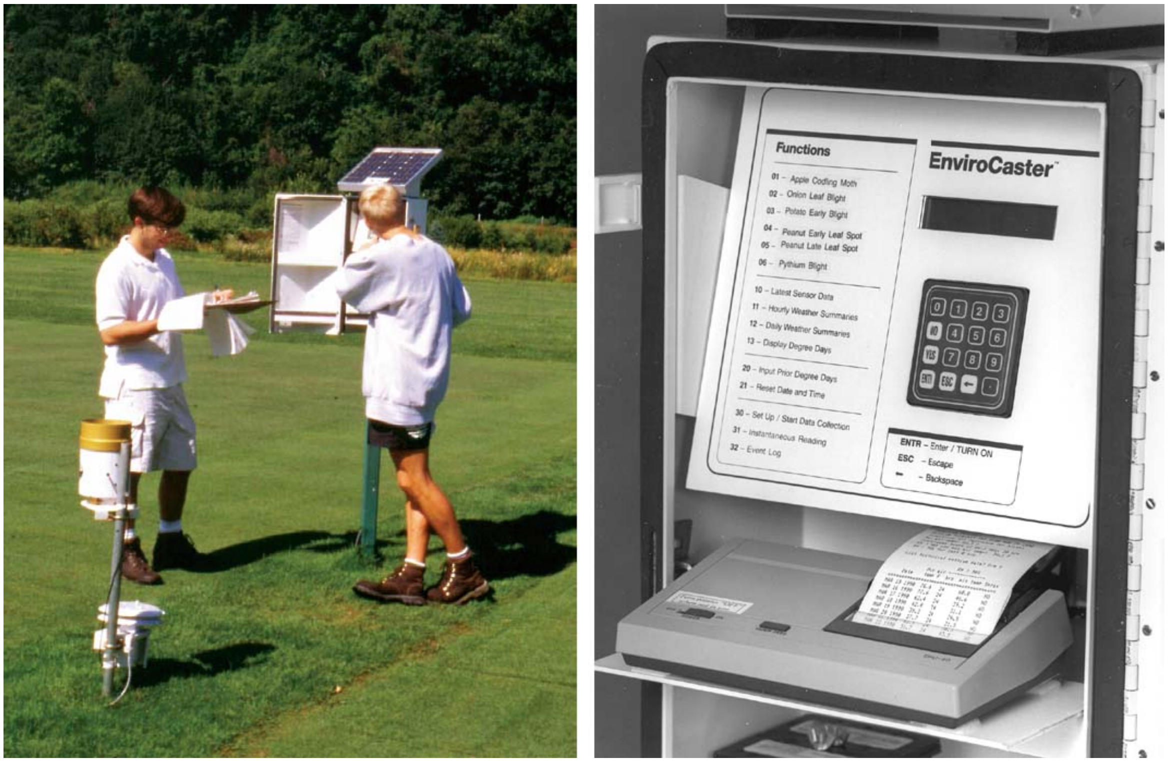

Functions
01 – Apple Codling Moth
02 – Onion Leaf Blight
03 – Potato Early Blight
04 – Peanut Early Leaf Spot
05 – Peanut Late Leaf Spot
06 – Pythium Blight
10 – Latest Sensor Data
11 – Hourly Weather Summaries
12 – Daily Weather Summaries
13 – Display Degree Days
20 – Input Prior Degree Days
21 – Reset Date and Time
30 – Set Up / Start Data Collection
31 – Instantaneous Reading
32 – Event Log
EnviroCaster™
ENTR – Enter / TURN ON
ESC – Escape
← – Backspace

Figure 10.9.
Computerized environmental-monitoring equipment can be used with models that predict disease outbreaks, based on environmental data for turfgrass and various other crops.

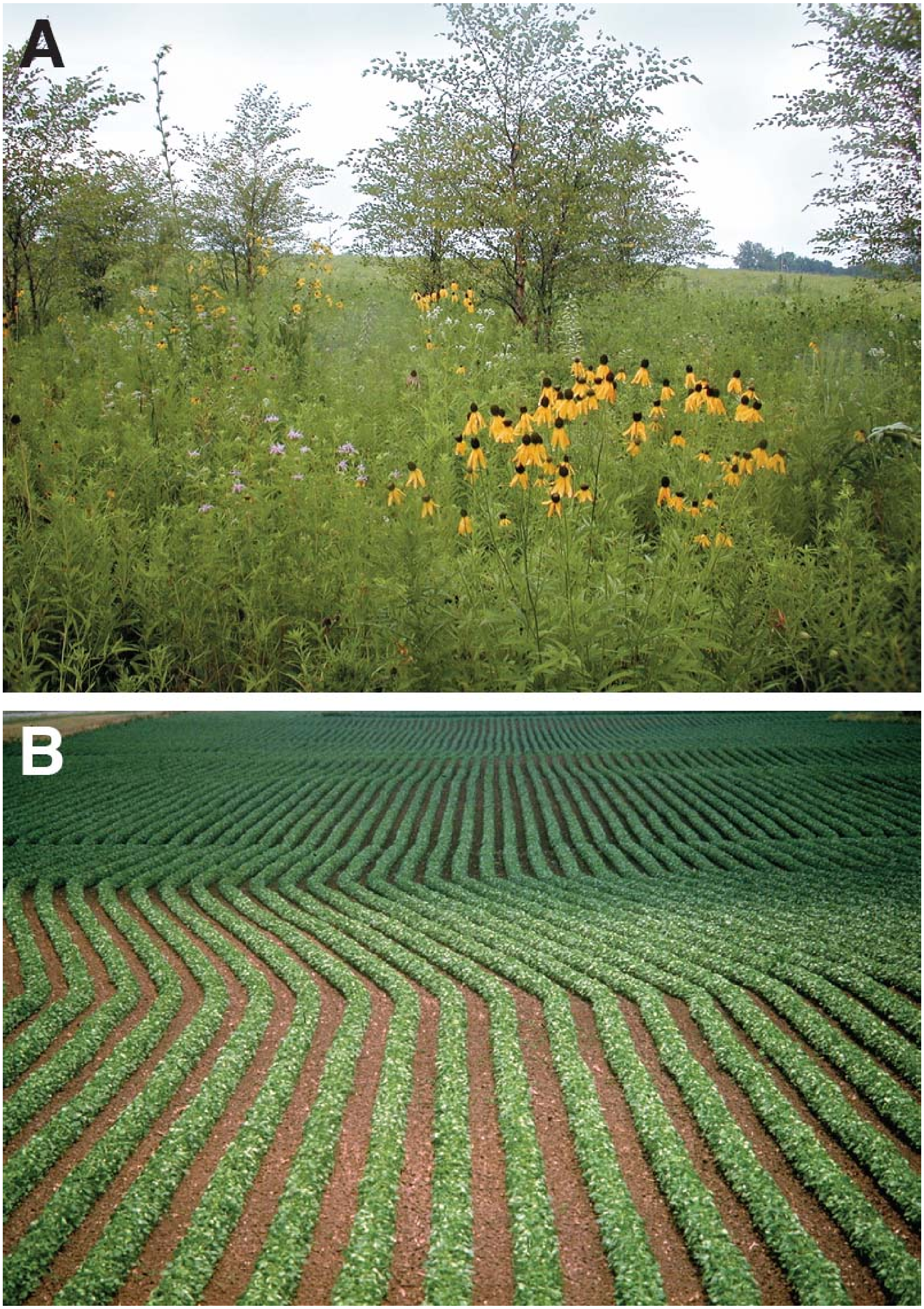
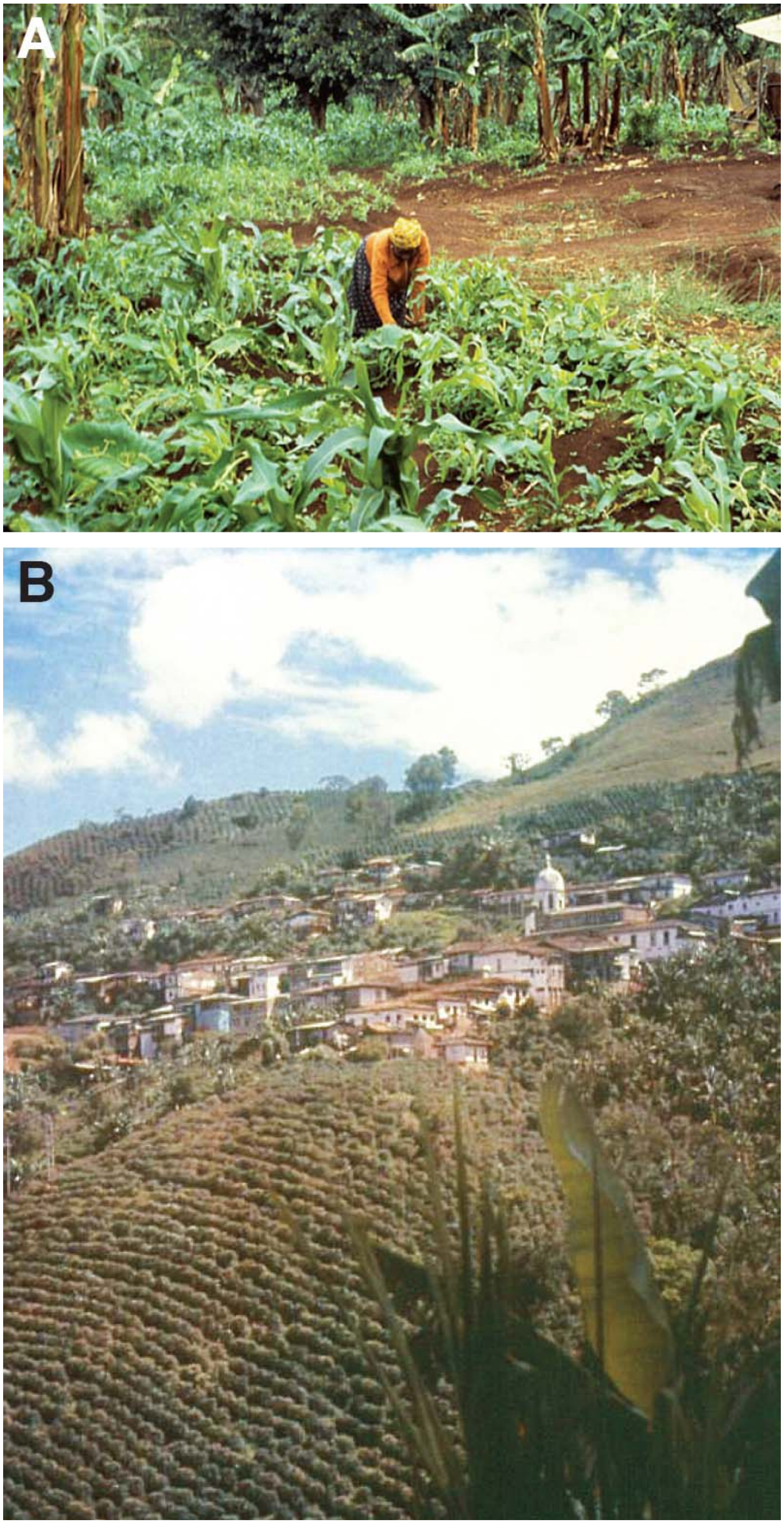

A
B

Figure 10.10.
Ecosystems of the U.S. Midwest. **A**, Natural ecosystem: a tall-grass prairie. **B**, Agro-ecosystem: a soybean field.


A
B

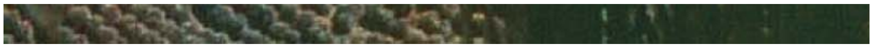


Figure 10.11.
A, Polyculture: mixed cropping (seven different crops) in Africa.
B, Monoculture: a coffee plantation in Colombia.

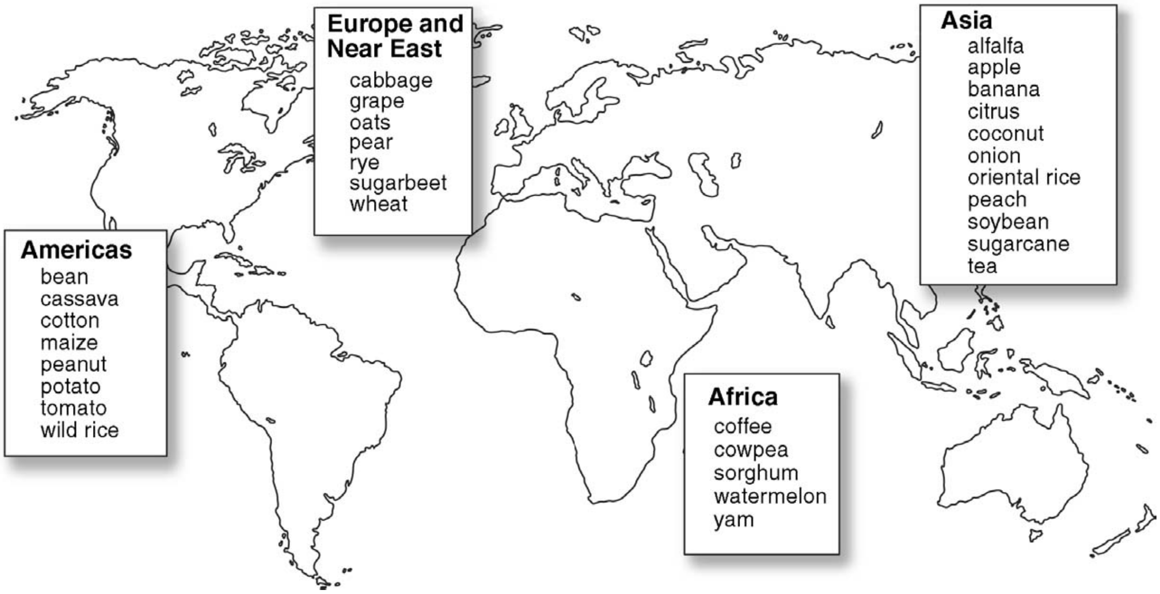

Europe and Near East
cabbage
grape
oats
pear
rye
sugarbeet
wheat
Asia
alfalfa
apple
banana
citrus
coconut
onion
oriental rice
peach
soybean
sugarcane
tea
Americas
bean
cassava
cotton
maize
peanut
potato
tomato
wild rice
Africa
coffee
cowpea
sorghum
watermelon
yam

Figure 10.12.
A map of sites of early plant domestication reveals how people have moved many important crops around the world.

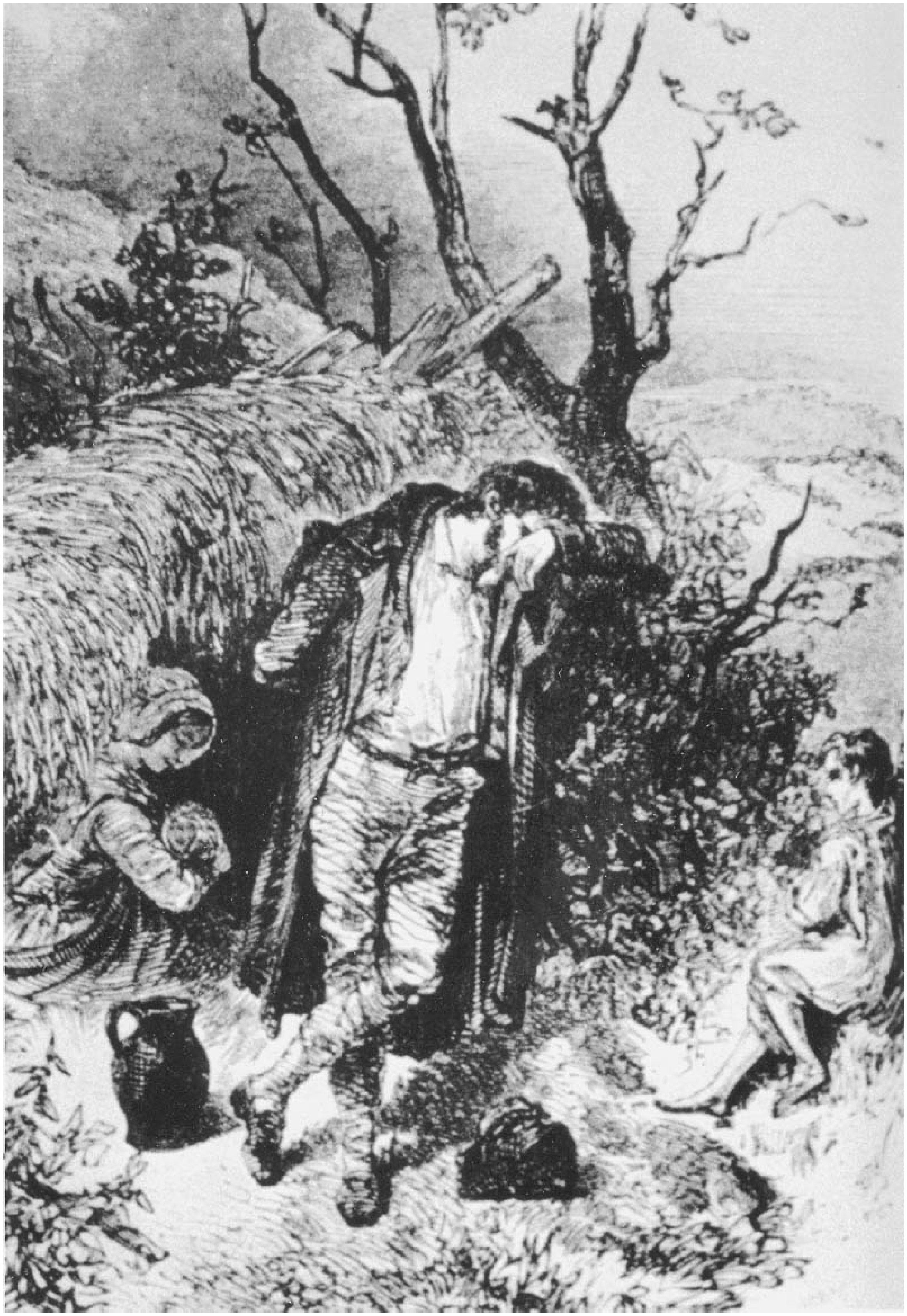


Figure 10.13.
“After the Eviction.” An Irish family evicted from their home because of failure to pay rent during the potato famine of the mid-1800s.

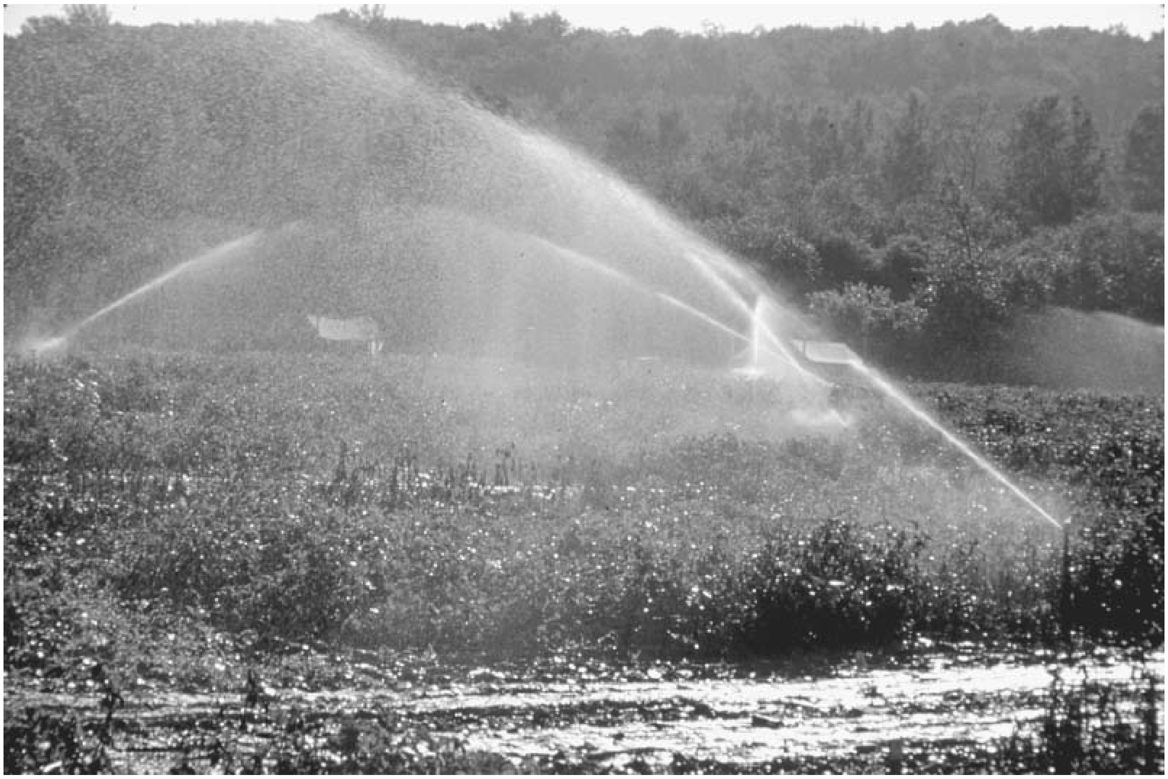


Figure 10.14.
Irrigation can cause prolonged leaf wetness, which increases many diseases.

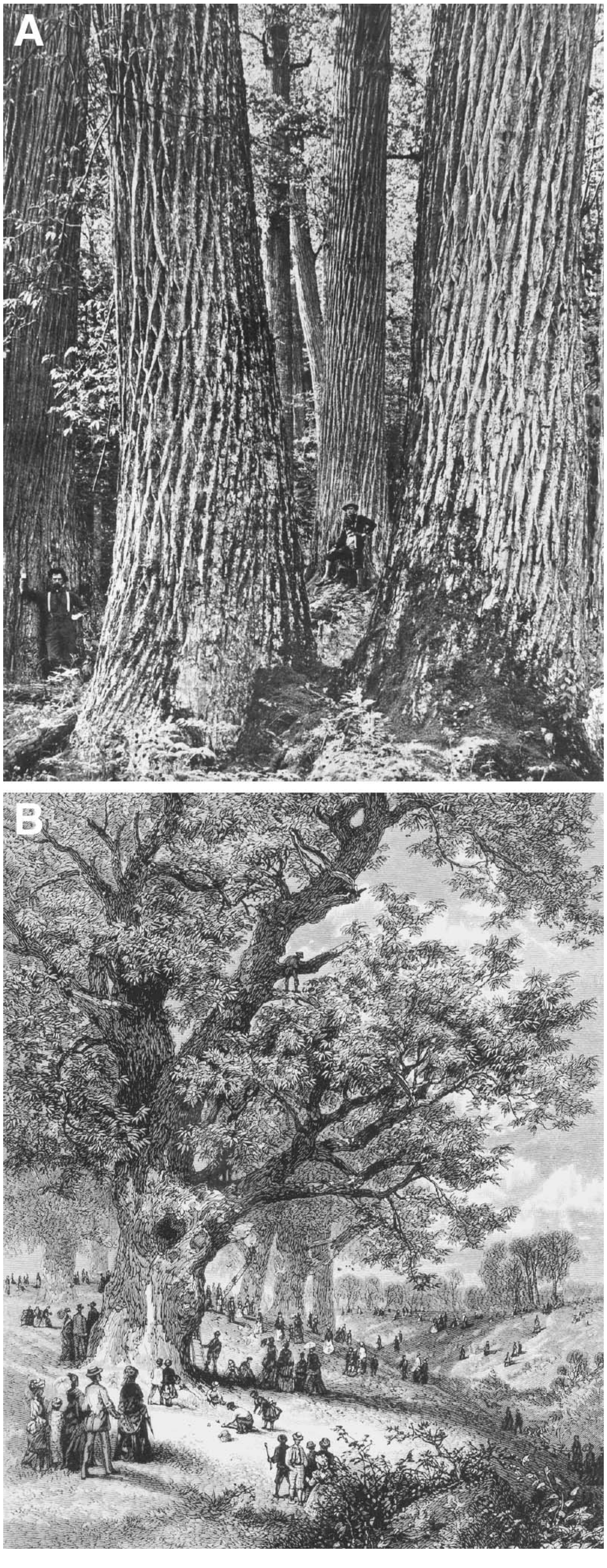

A
B

Figure 10.15.
Chestnut trees in **(A)** the Appalachian forest and **(B)** a park in Philadelphia before chestnut blight had occurred in the United States.

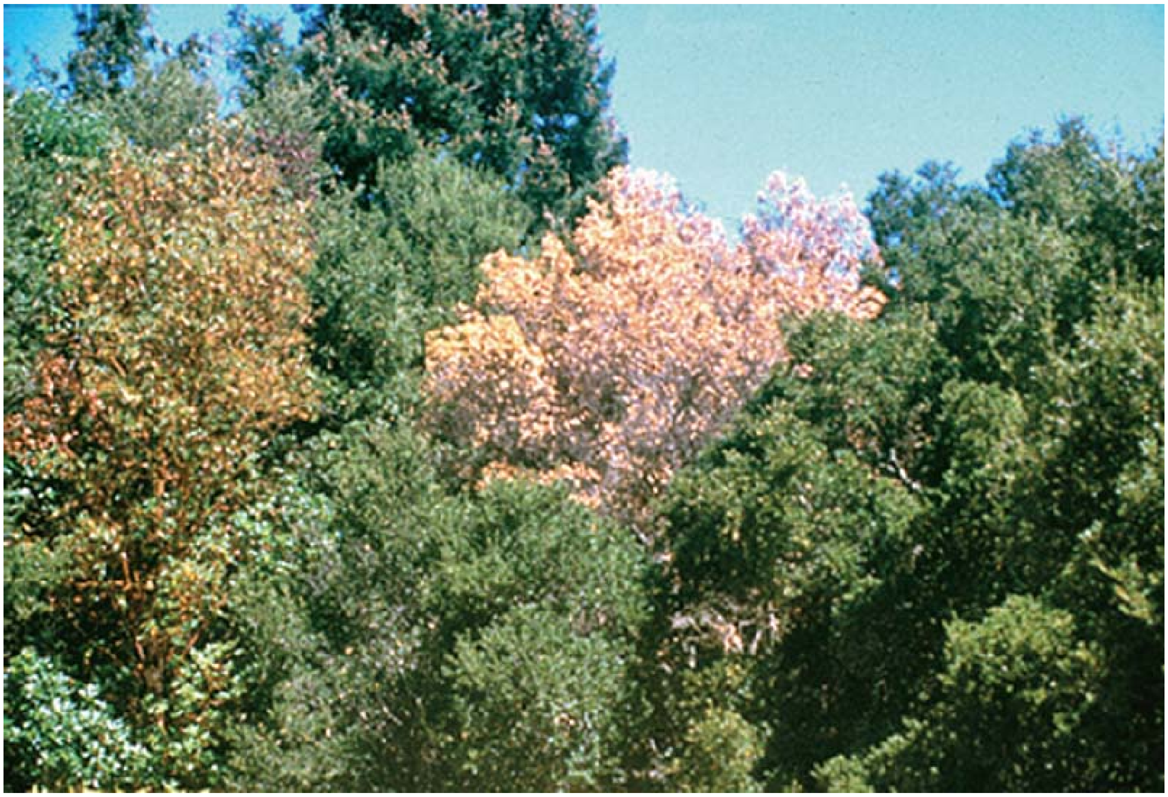


Figure 10.16.
Ramorum blight, also known as sudden oak death, caused by *Phytophthora ramorum*, in California.

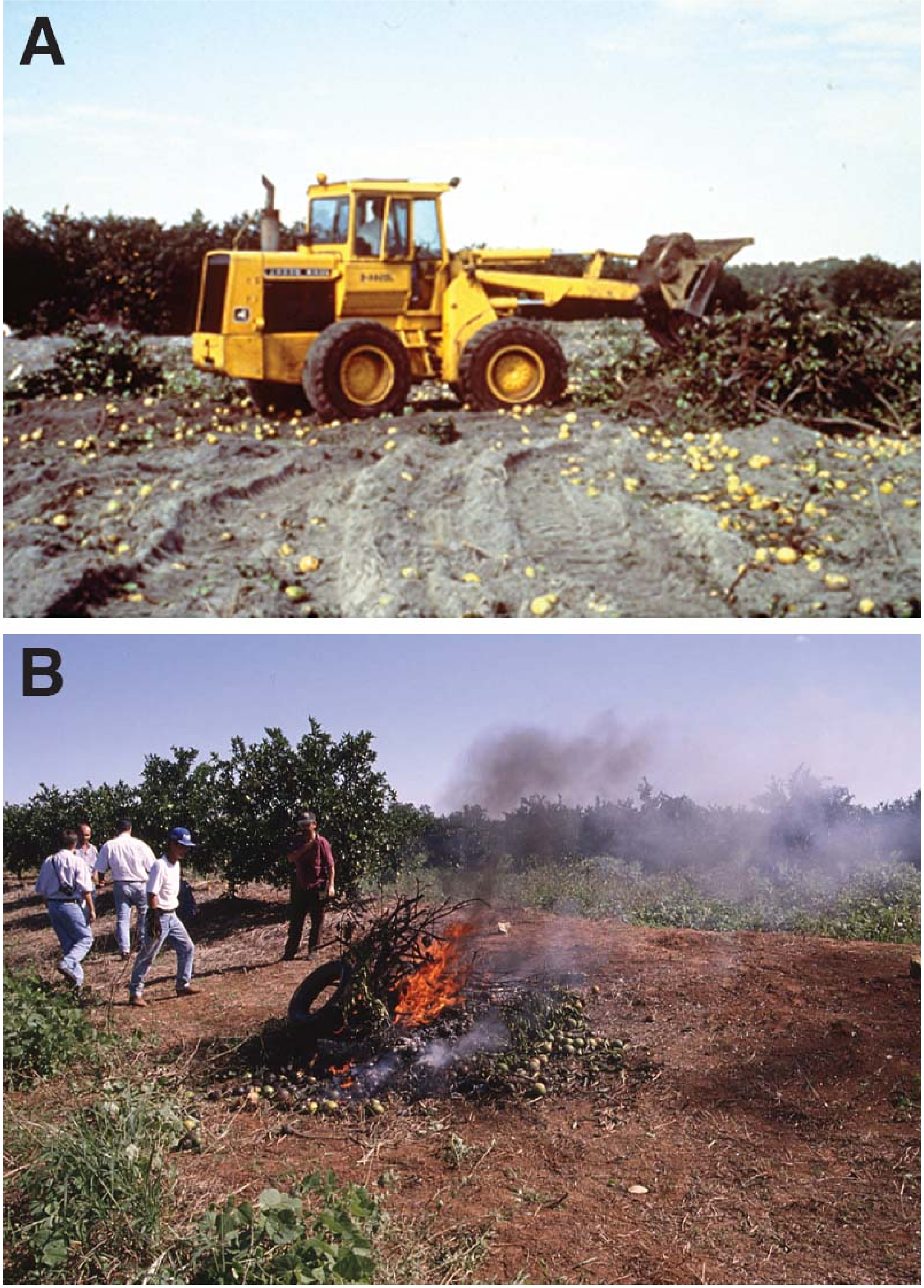


Figure 10.17.

Citrus trees infected with citrus canker destroyed by plowing (**A**) and burning (**B**).

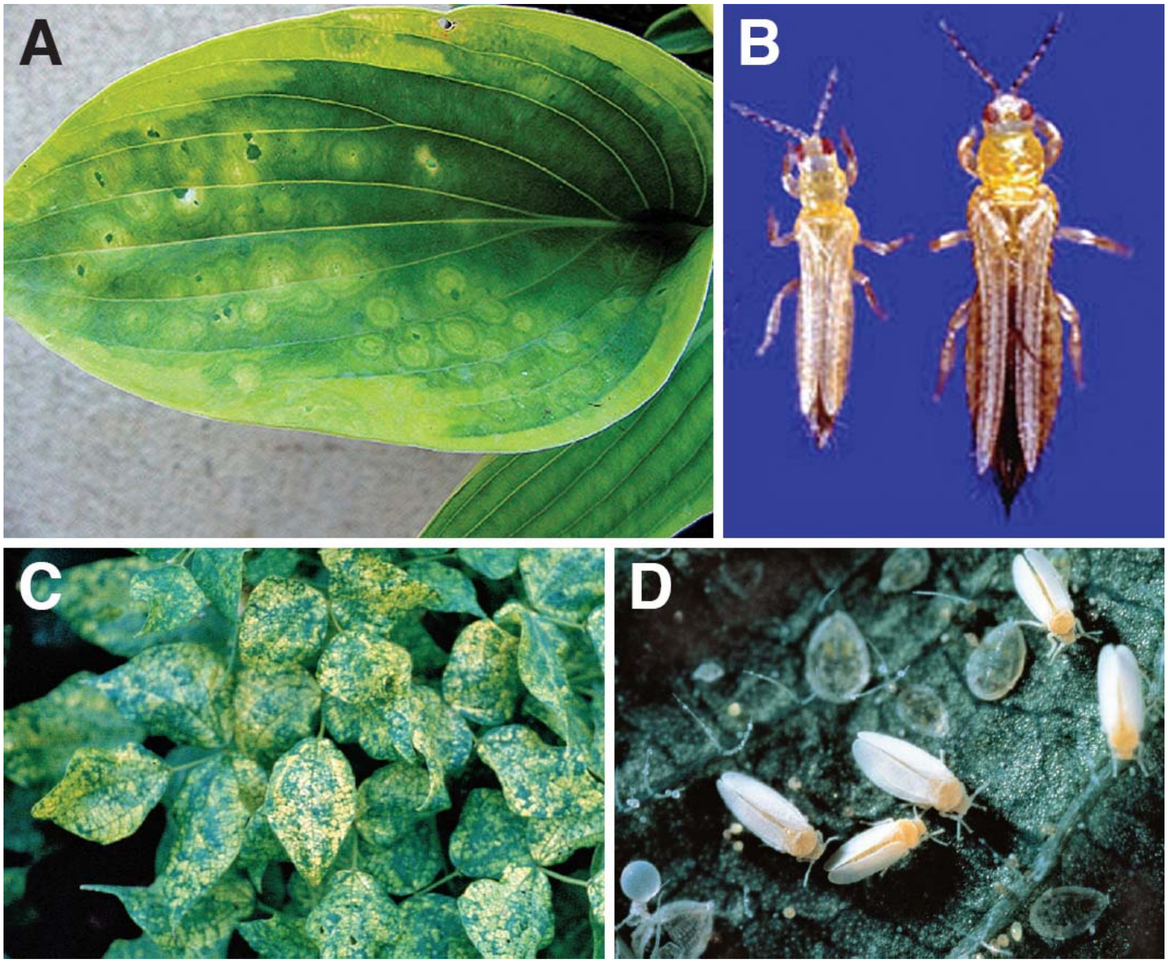


Figure 10.18.
Tospoviruses, such as *Tomato spotted wilt virus*, which causes a disease of hosta (**A**), are transmitted by thrips (**B**). Some geminiviruses, such as *Bean golden mosaic virus* (**C**), are transmitted by whiteflies (**D**).

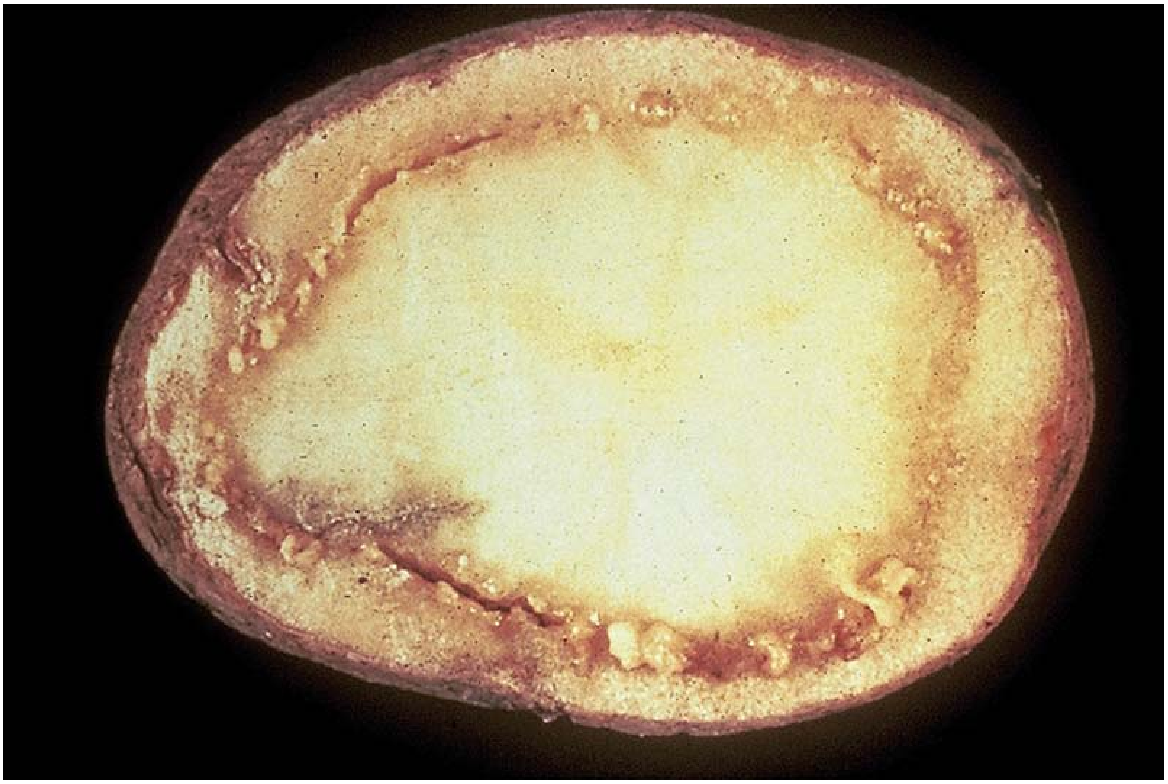


Figure 10.19.
Ring rot of potato. Cutting tools can easily spread the bacterial ooze from a diseased tuber to the cut surfaces of seed tubers.

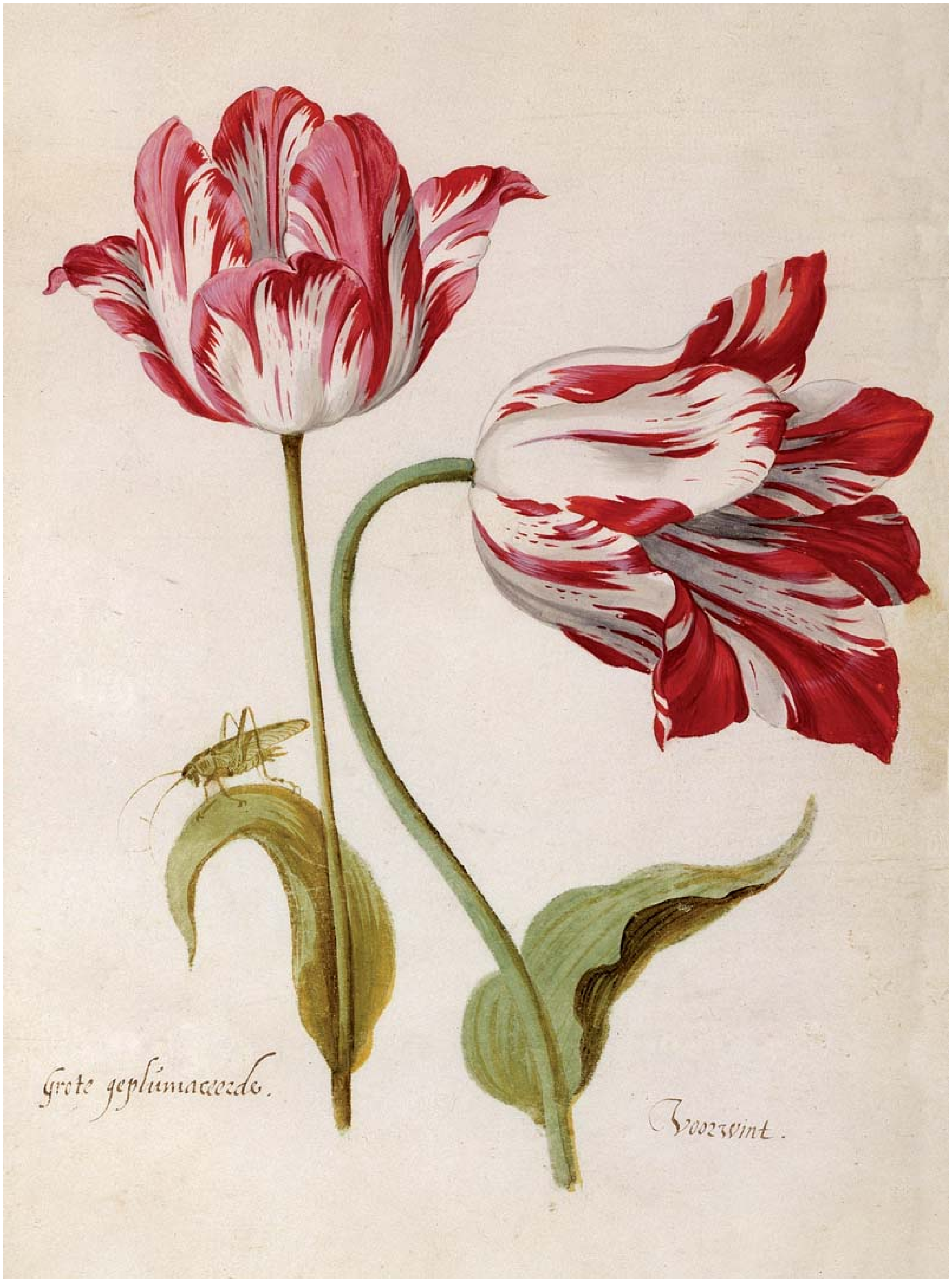

Grote geplumaceerde.
Voorwint.

Figure 10.20.

Tulips with color break in a painting by Dutch artist Jakob Marrel (1614-1681).

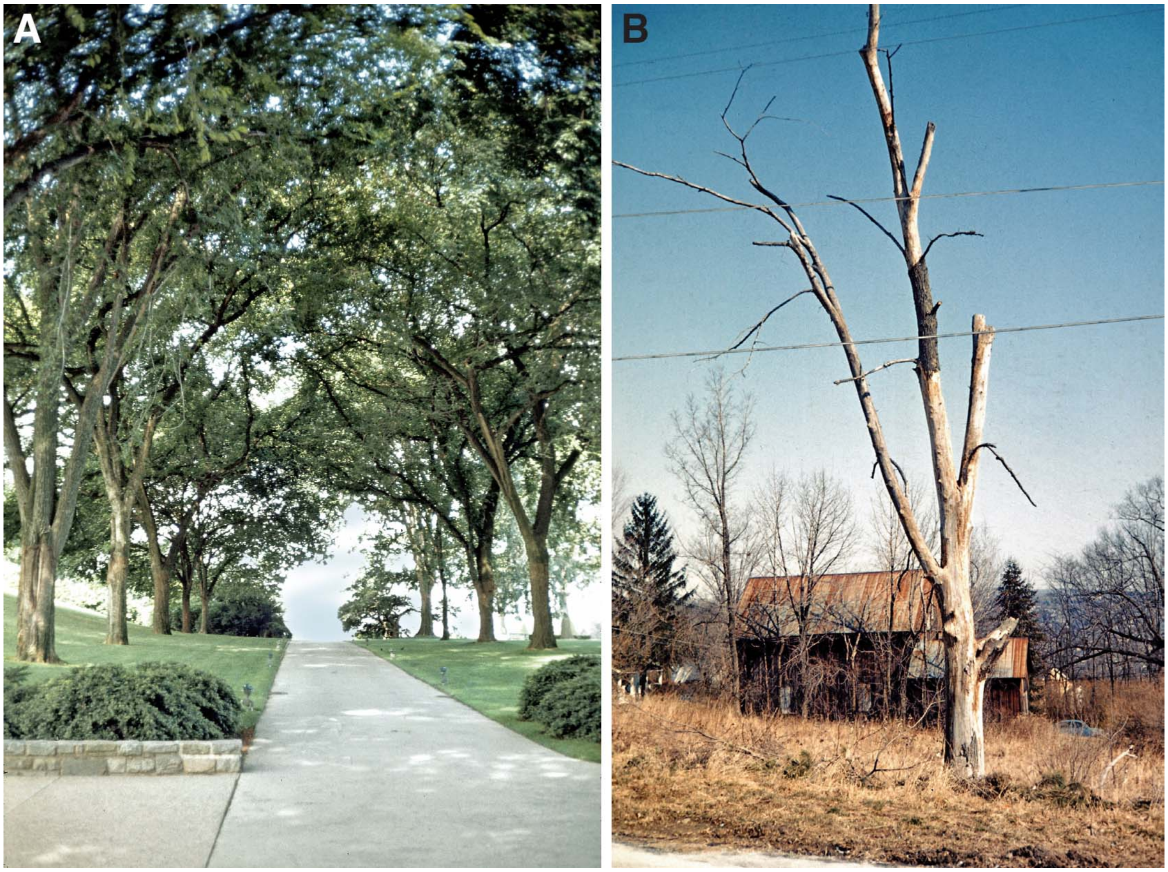


Figure 10.21.
American elms (**A**) lined many streets in the United States before they were killed by Dutch elm disease (**B**).

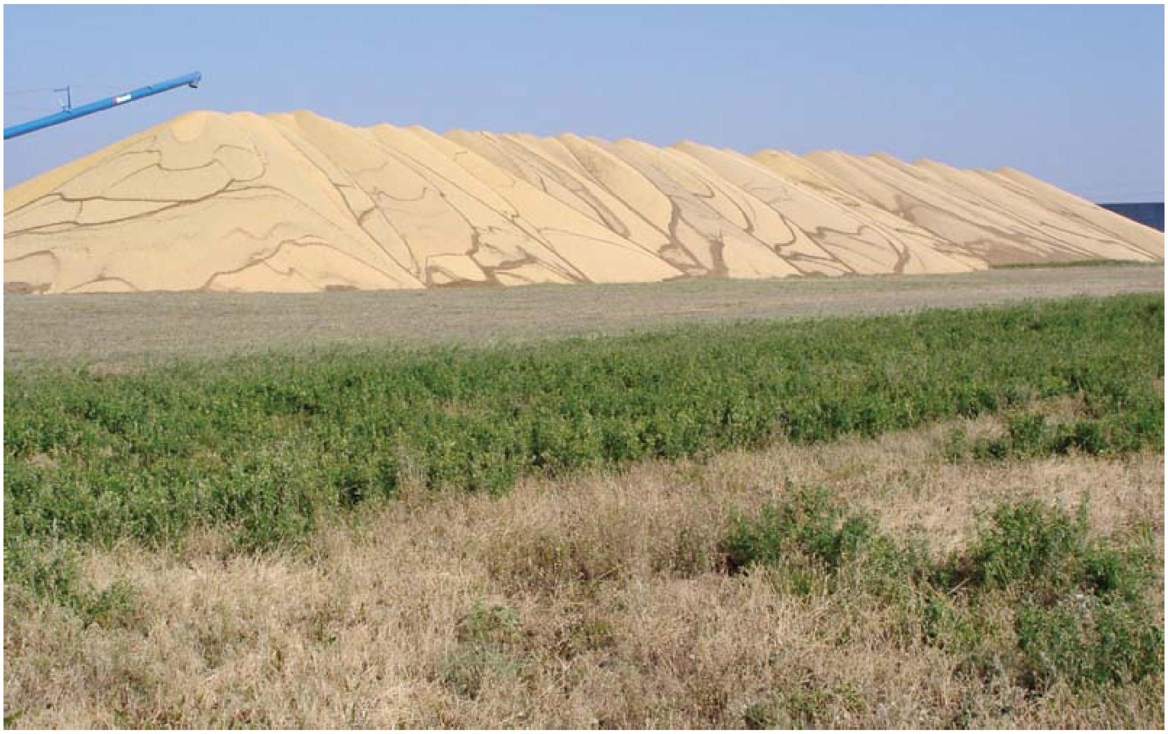


Figure 10.22.
Large quantities of food and feed (soybeans are shown here) must be stored after harvest.

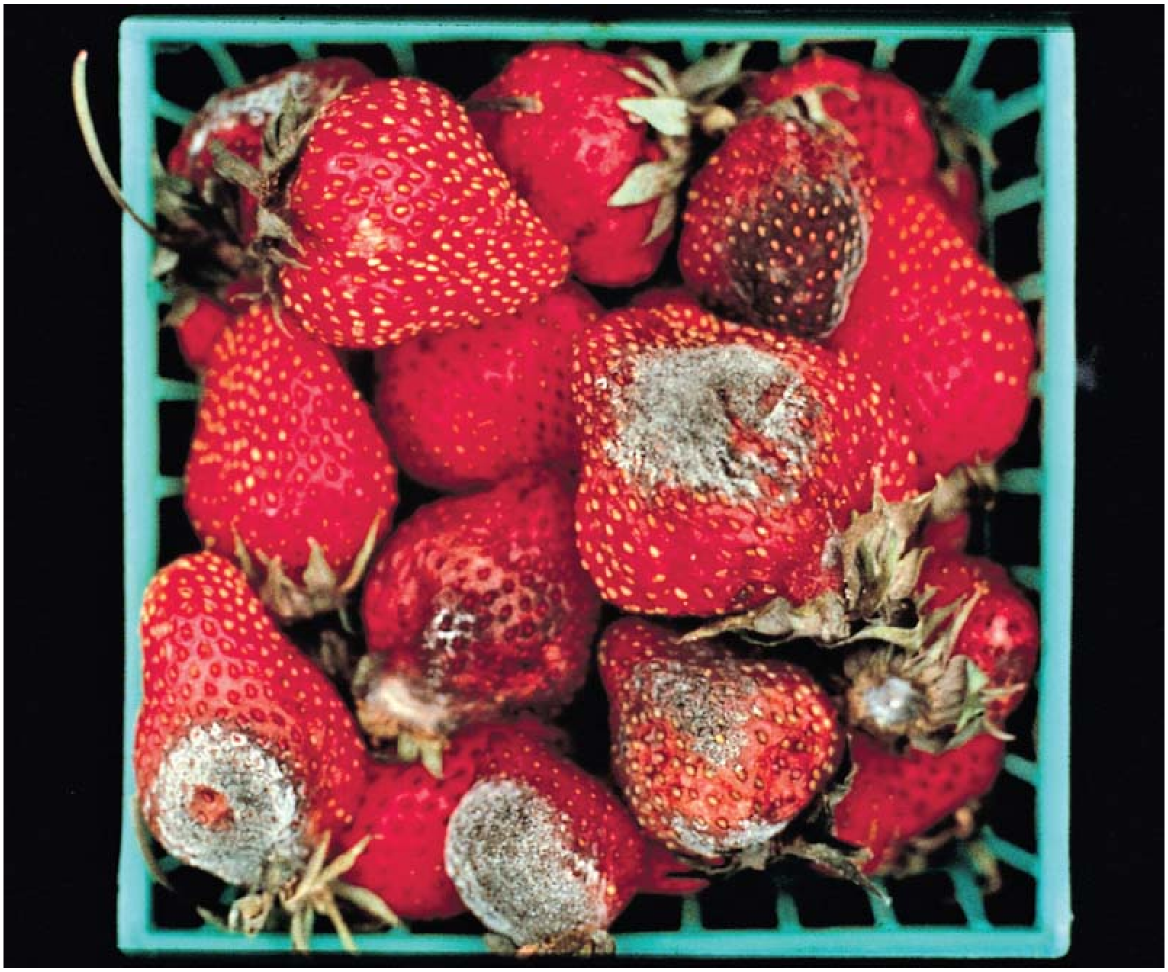


Figure 10.23.
Strawberries that have rotted after harvest as a result of gray mold, caused by *Botrytis cinerea*.

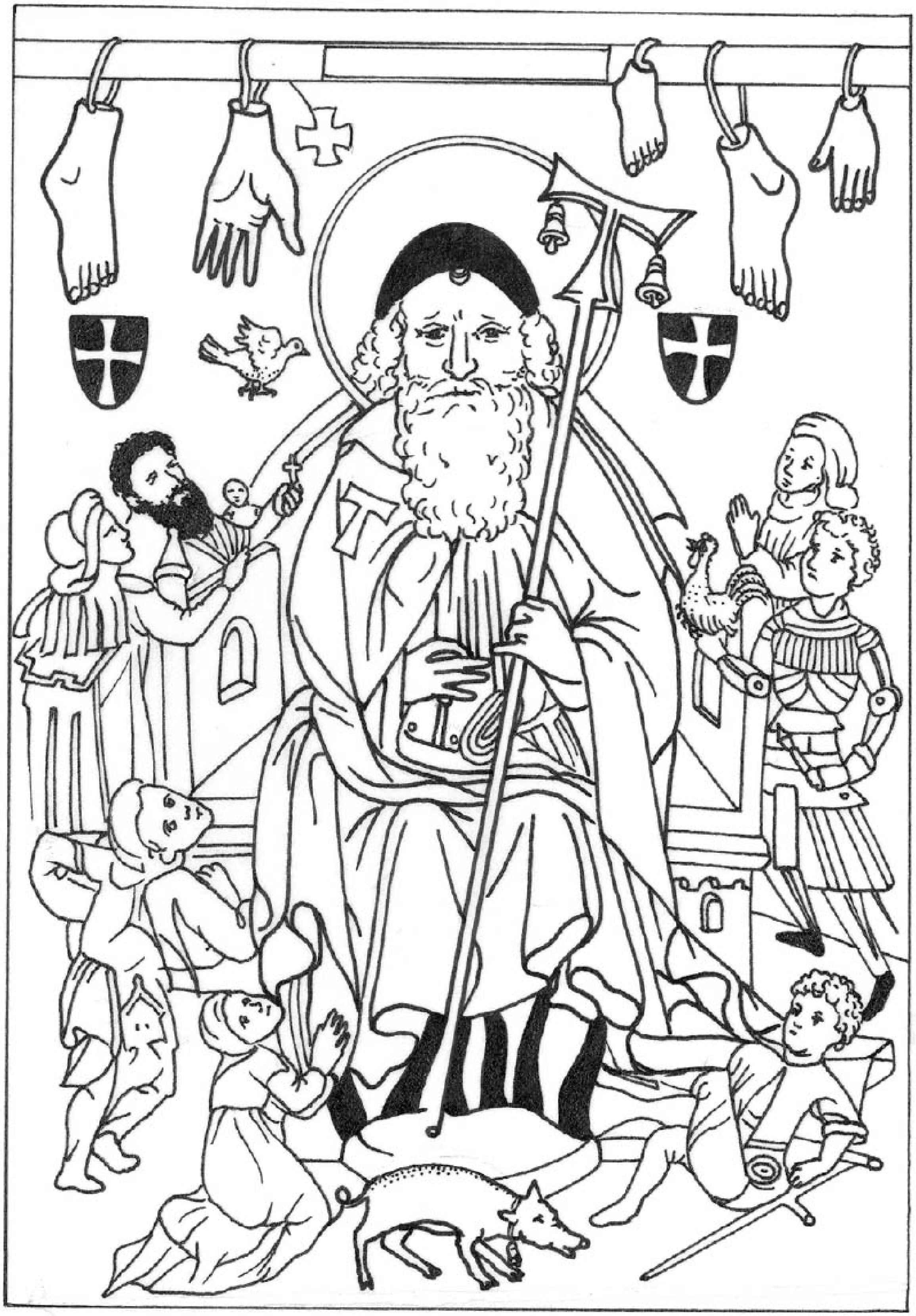


Figure 10.24.
St. Anthony. Redrawn from a woodcut made in Germany about 1215. Note the hands and feet hanging on the top bar. Loss of extremities from gangrene was an important symptom of ergotism, also known as St. Anthony's fire, a disease caused by mycotoxin poisoning.

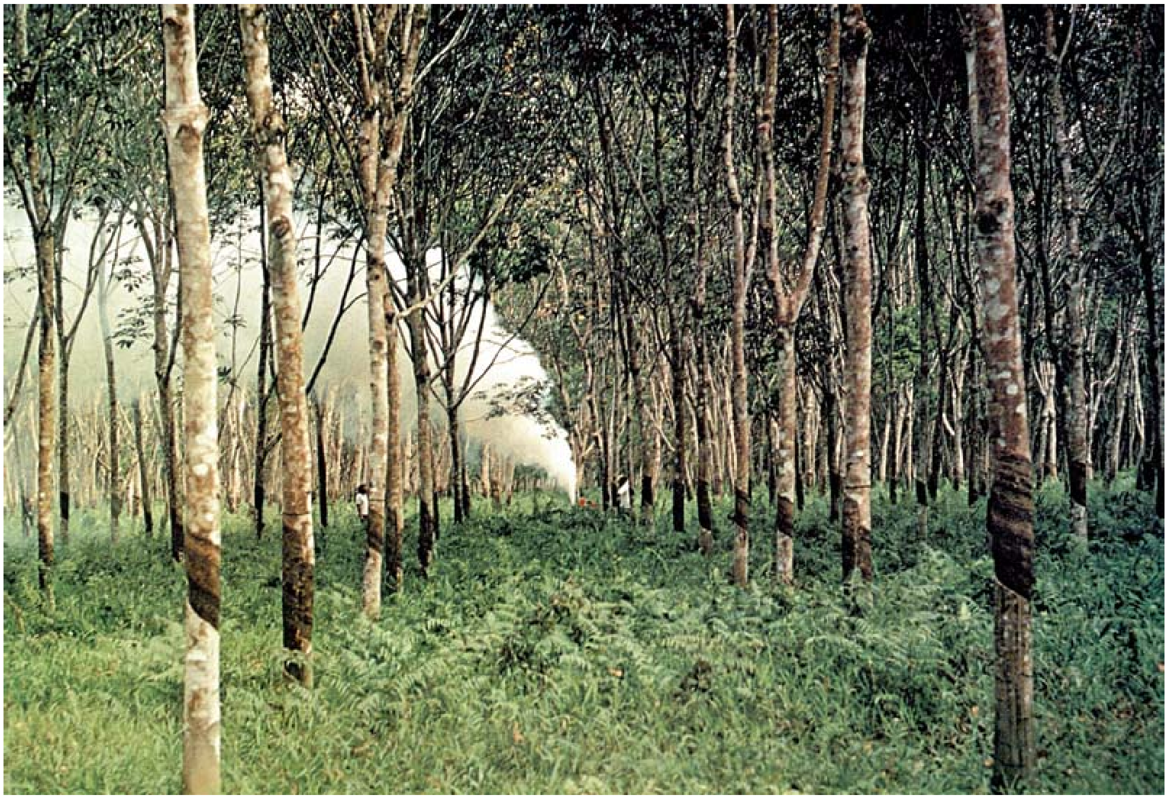


Figure 11.1.
Rubber plantation in the Philippines.

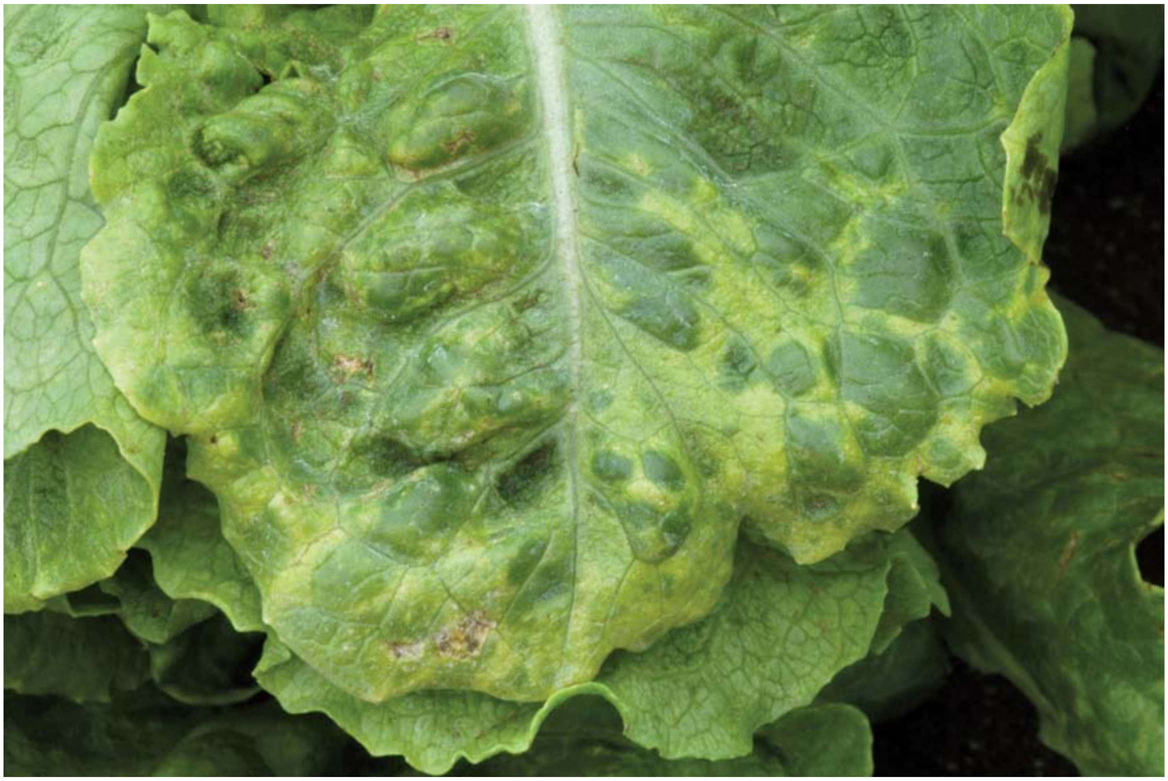


Figure 11.2.
Romaine lettuce infected by *Lettuce mosaic virus*.

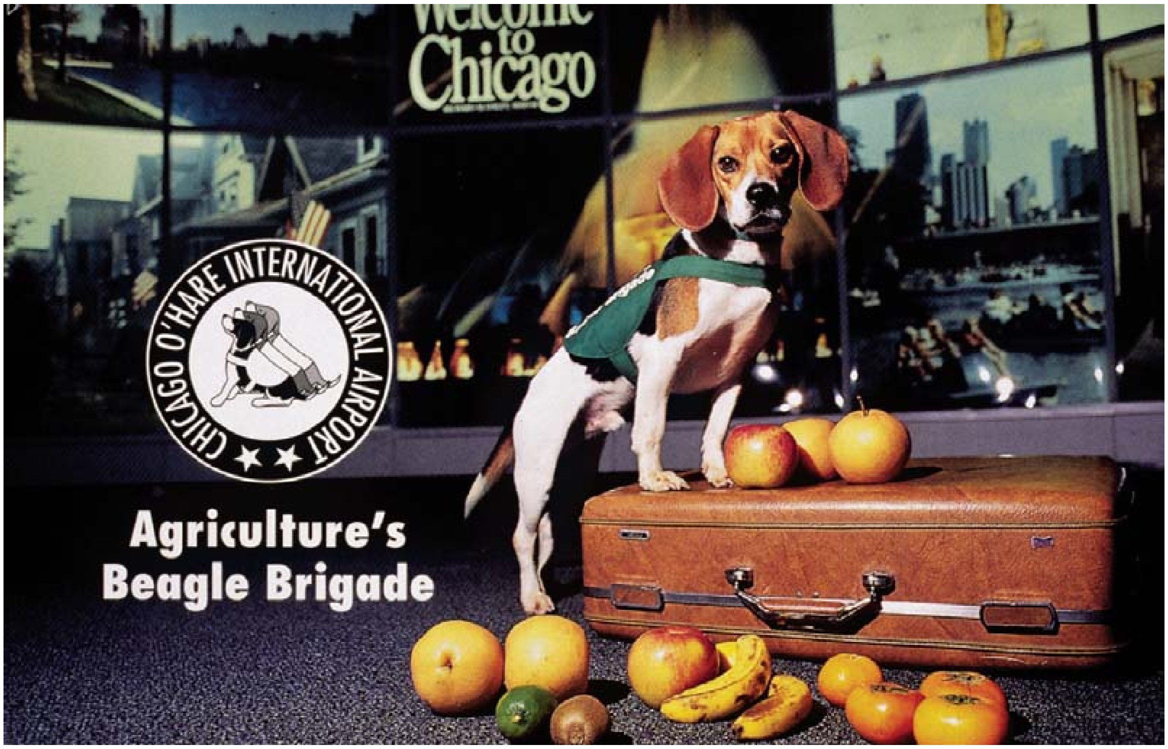

Welcome to Chicago
CHICAGO O'HARE INTERNATIONAL AIRPORT
Agriculture's Beagle Brigade

Figure 11.3.
Beagles are trained to help detect illegal fruits and vegetables at ports of entry.

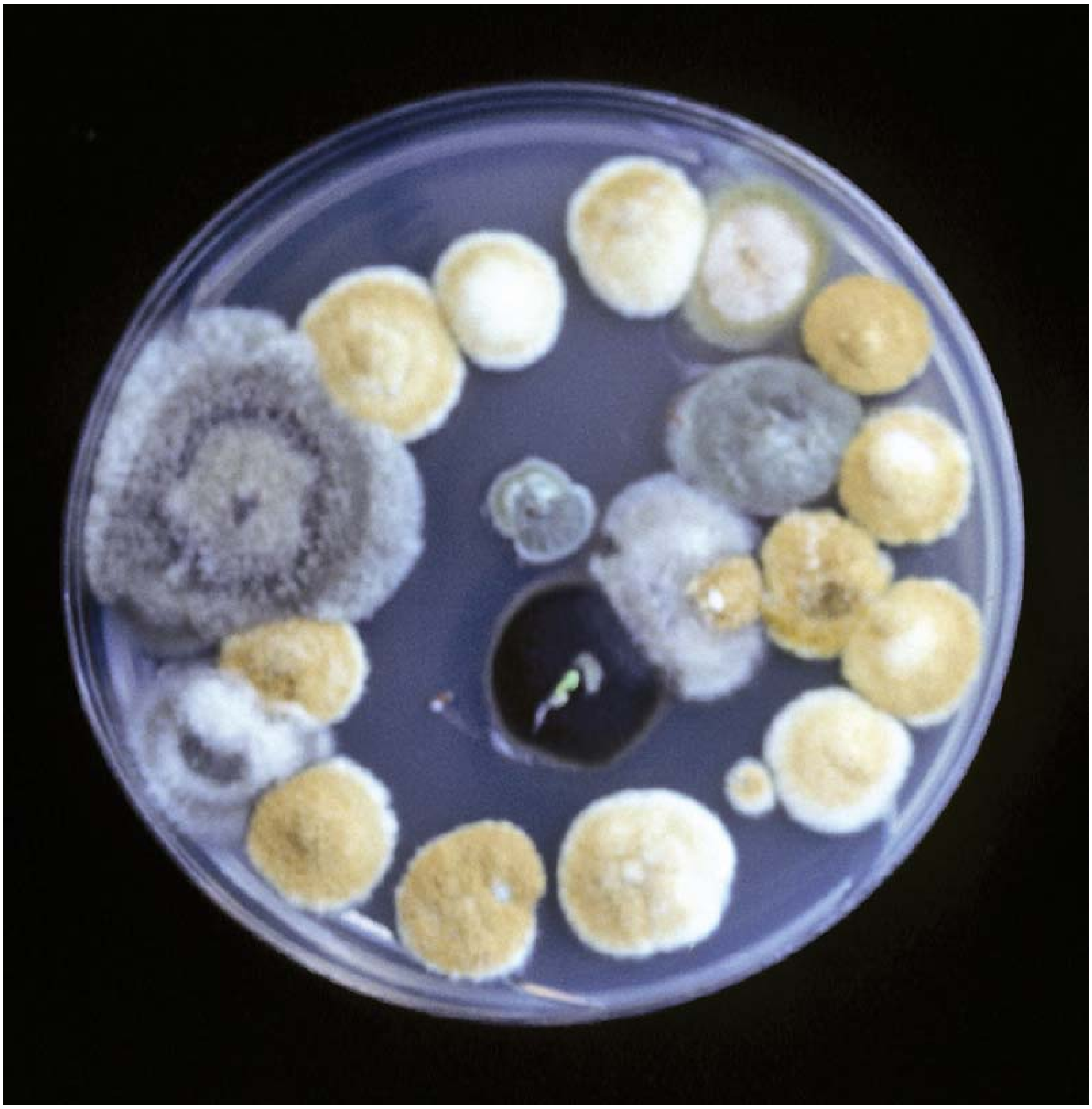


Figure 11.4.
A variety of fungal contaminants growing from infested seed placed on a nutrient medium.

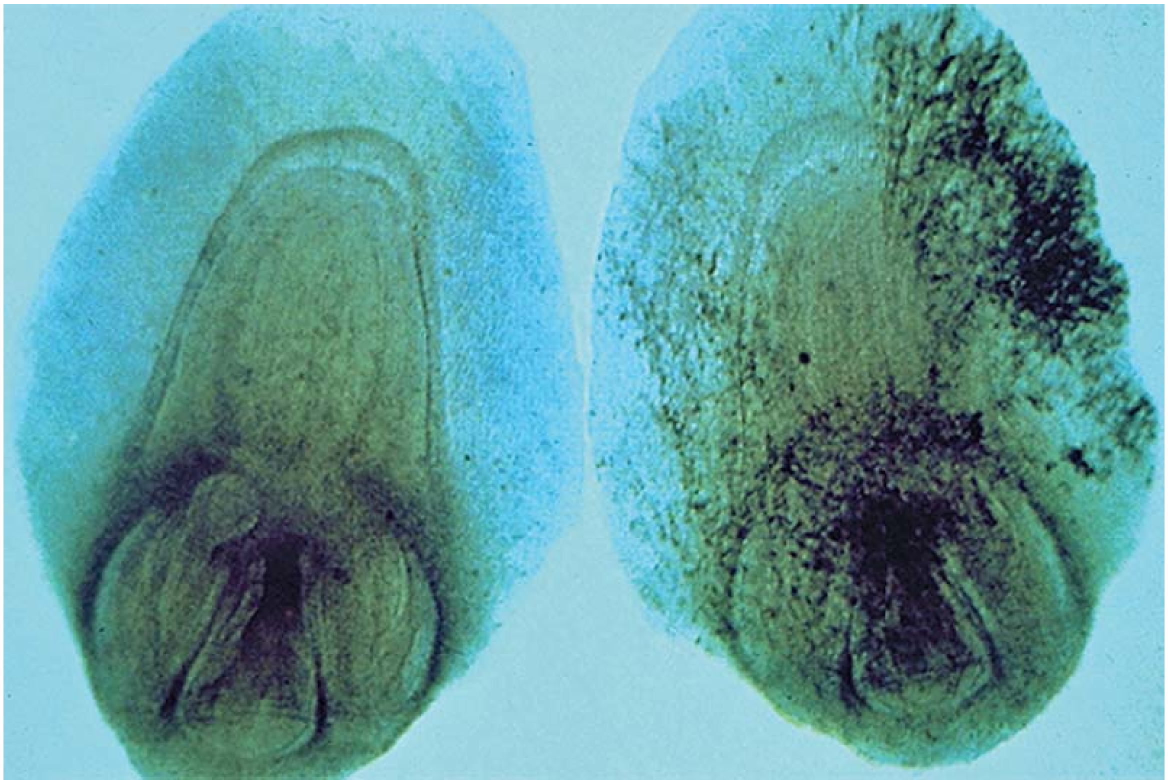


Figure 11.5.
Barley seed embryo infected by the loose smut pathogen (*Ustilago nuda*) (right) and an uninfected embryo (left).

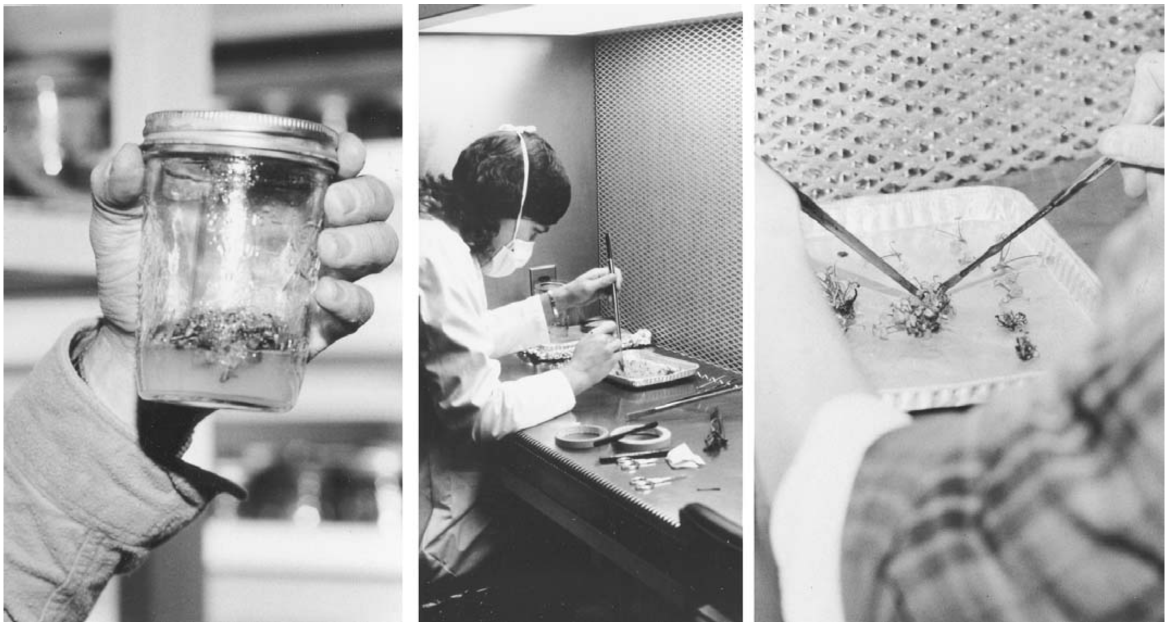
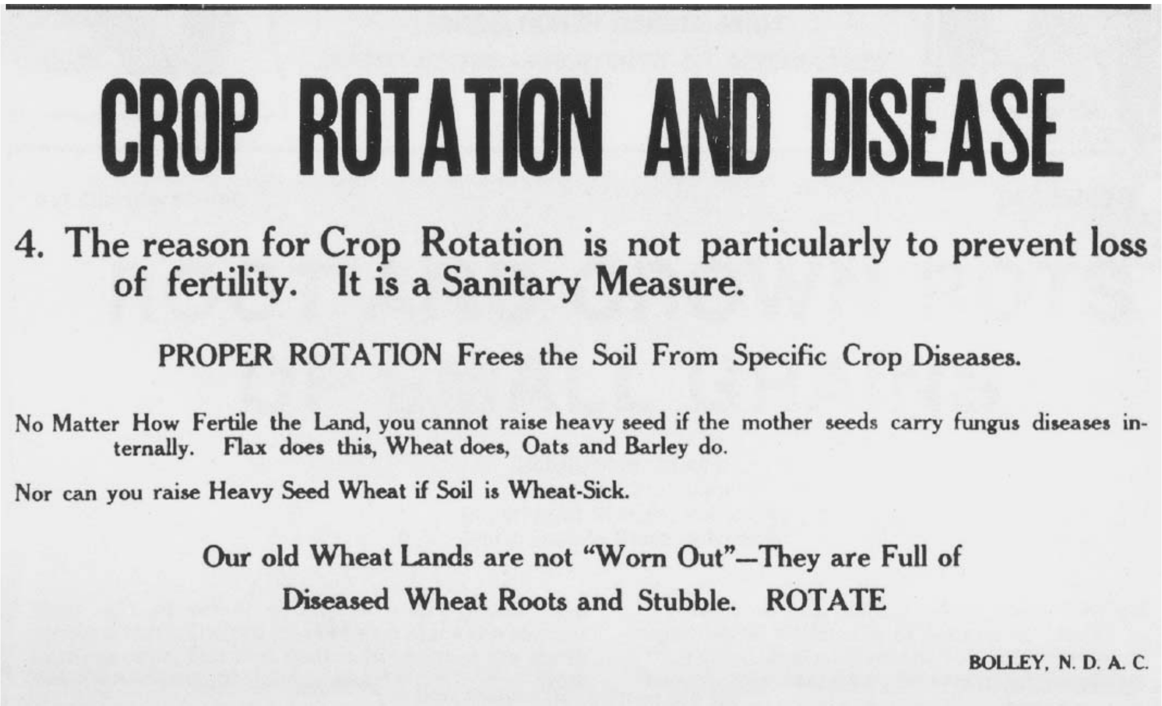


Figure 11.6.
Regeneration of plants from a meristem. **Left**, Growth of the meristem tissue on a nutrient medium containing appropriate growth regulators to produce multiple shoots. **Center and right**, Under sterile conditions, the tiny shoots can be separated and grown into plants.



CROP ROTATION AND DISEASE

4. The reason for Crop Rotation is not particularly to prevent loss of fertility. It is a Sanitary Measure.

PROPER ROTATION Frees the Soil From Specific Crop Diseases.

No Matter How Fertile the Land, you cannot raise heavy seed if the mother seeds carry fungus diseases internally. Flax does this, Wheat does, Oats and Barley do.

Nor can you raise Heavy Seed Wheat if Soil is Wheat-Sick.

Our old Wheat Lands are not "Worn Out"—They are Full of Diseased Wheat Roots and Stubble. ROTATE

BOLLEY, N. D. A. C.

Figure 11.7.
A poster by H. L. Bolley, from North Dakota in 1909, explaining that the benefits of rotation of wheat with other crops result from a reduction of soilborne pathogens.

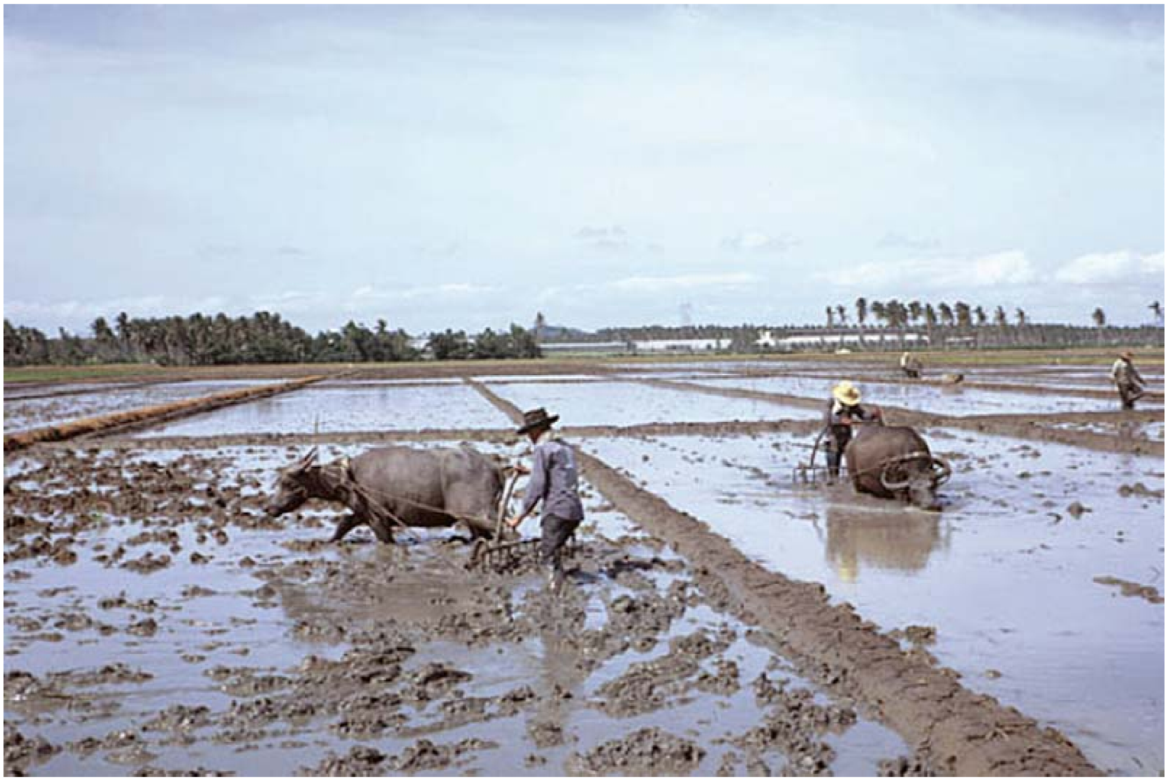


Figure 11.8.
Flooding and tillage before rice cultivation in the Philippines.

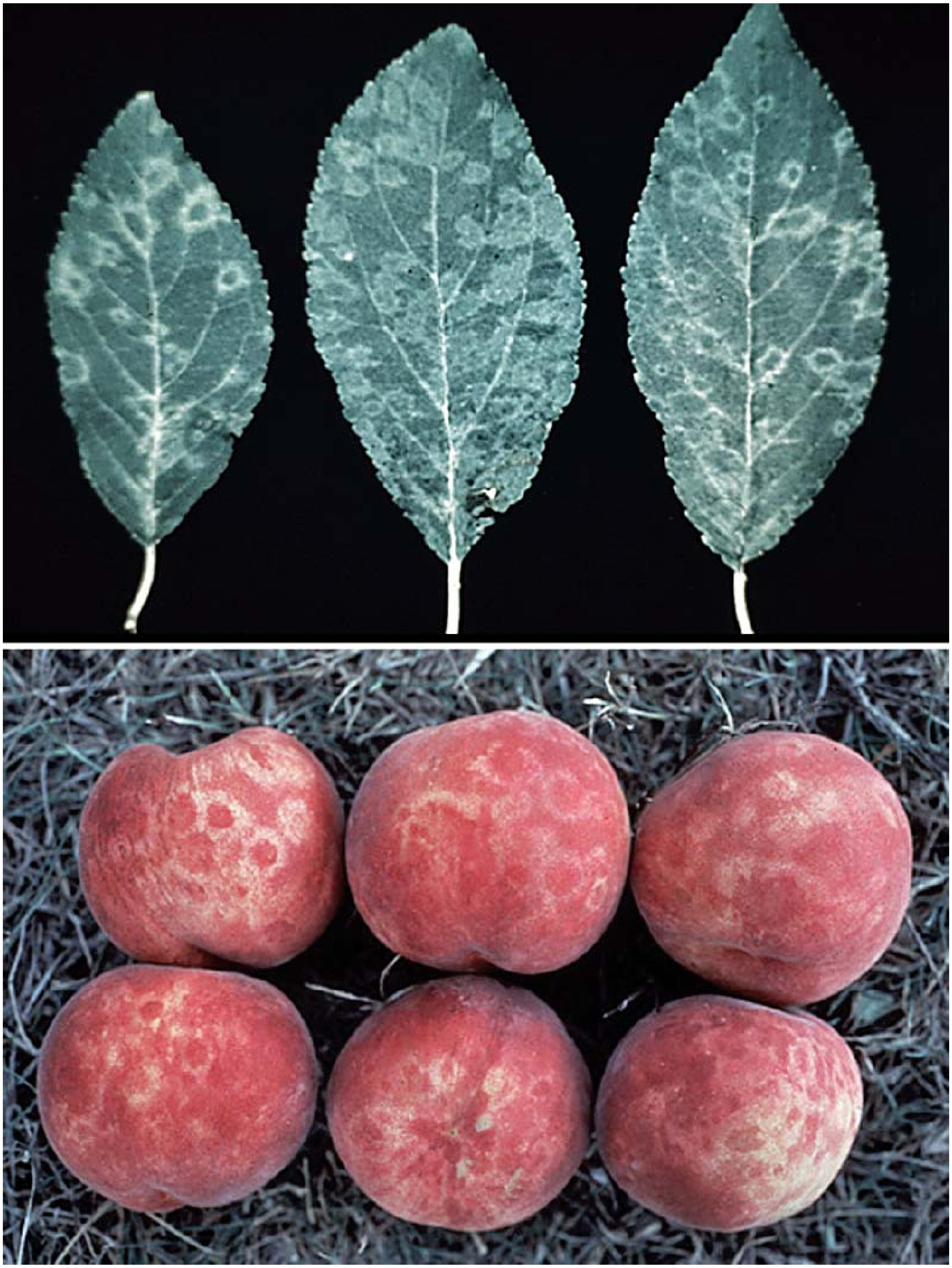


Figure 11.9.
Symptoms of *Plum pox virus* of stone fruits.

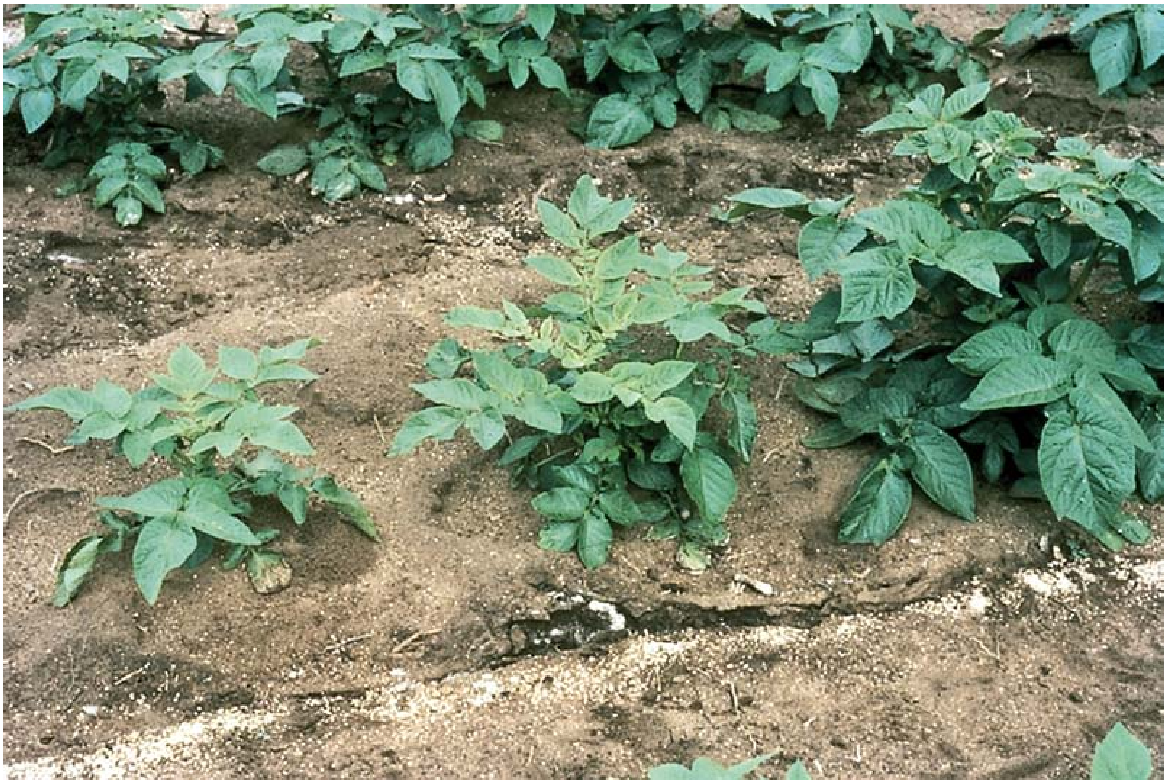


Figure 11.10.
Virus-infected potato plants should be removed to prevent the spread of the virus to healthy plants.

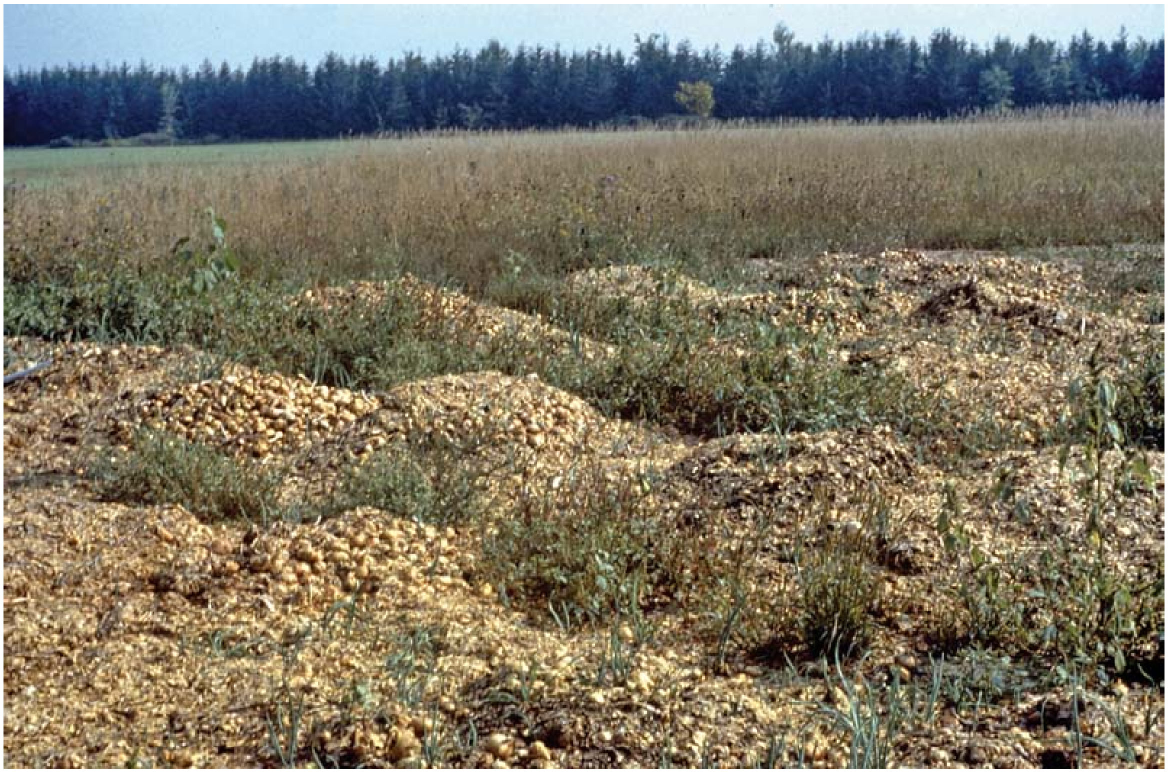


Figure 11.11.
Onion cull piles are a source of *Onion yellow dwarf virus*, which is spread by aphid vectors.

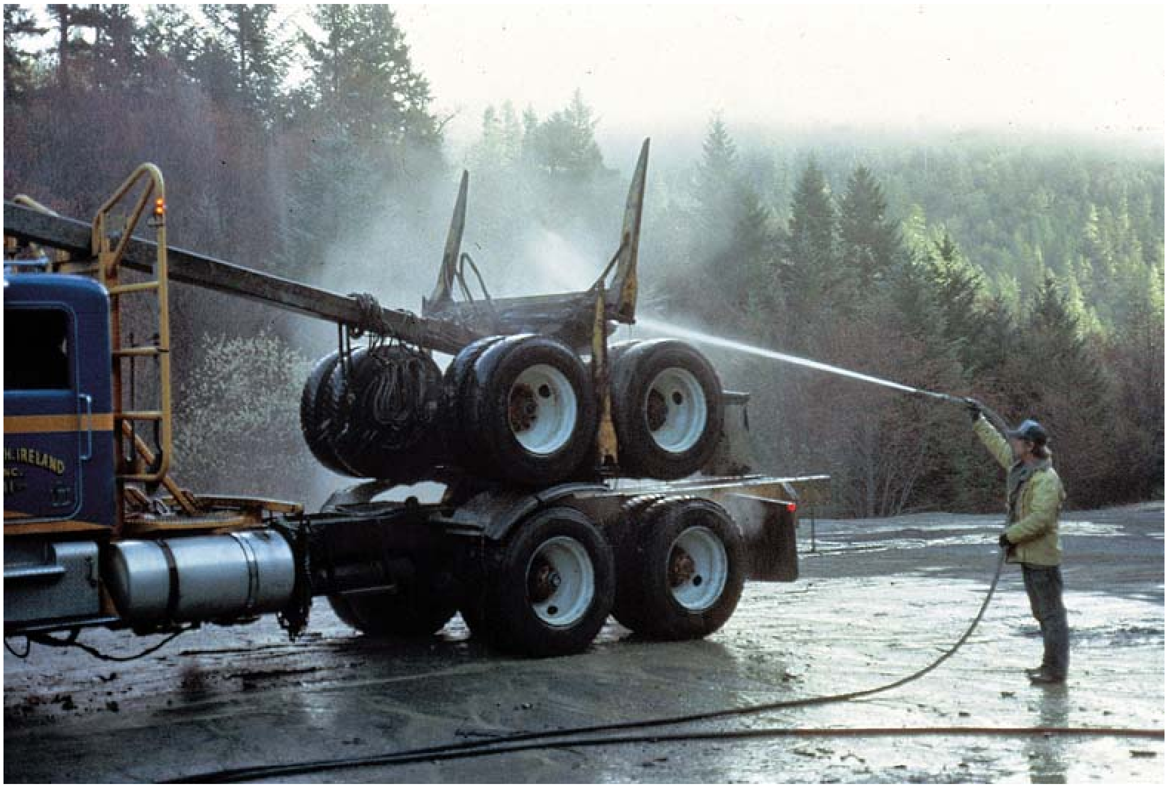


Figure 11.12.
Washing a log truck before it enters a “clean” area to prevent the spread of *Phytophthora*, a soilborne pathogen.

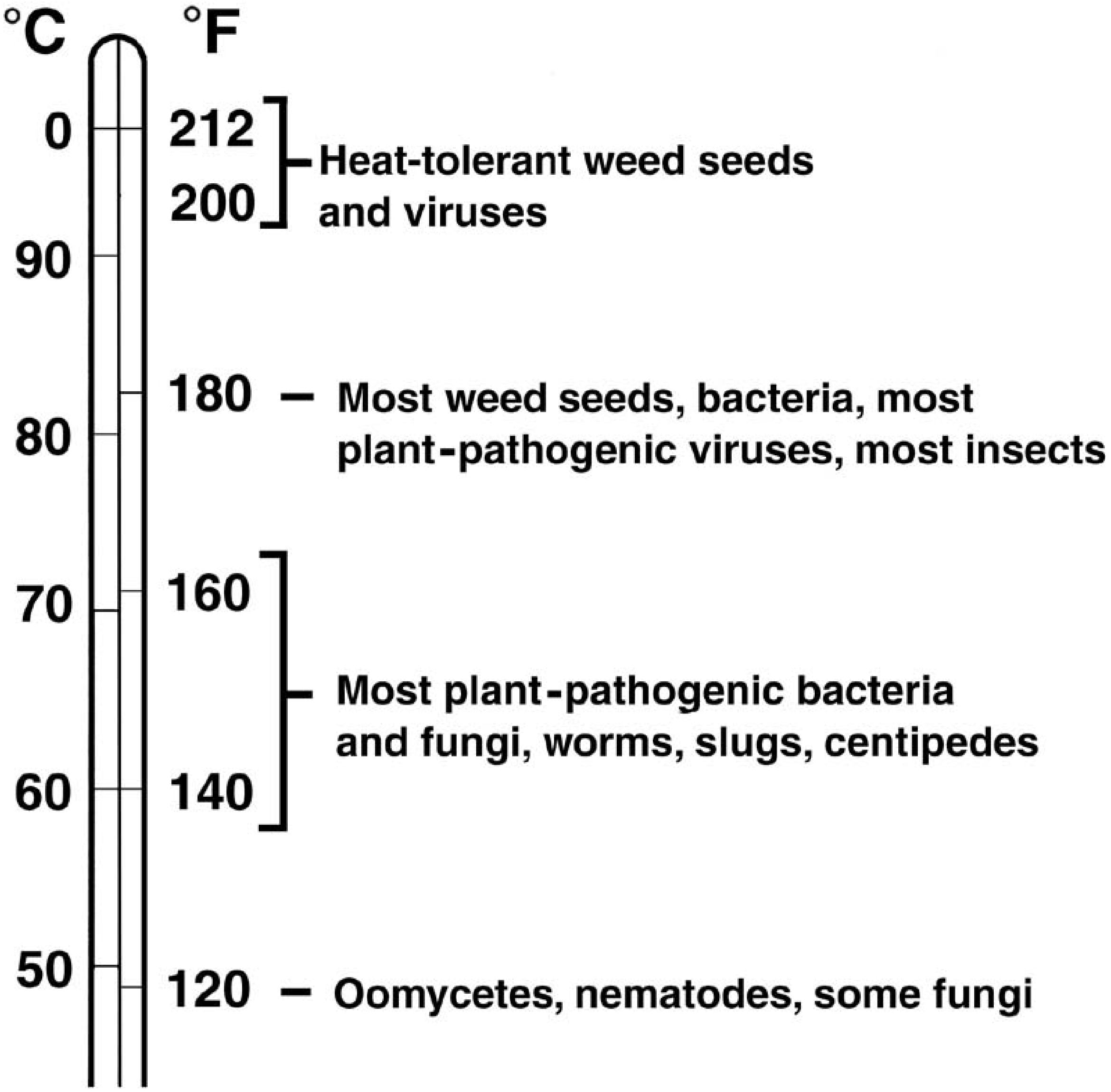

°C
°F
0
212
Heat-tolerant weed seeds and viruses
200
90
180
Most weed seeds, bacteria, most plant-pathogenic viruses, most insects
80
160
70
Most plant-pathogenic bacteria and fungi, worms, slugs, centipedes
60
140
50
120
Oomycetes, nematodes, some fungi

Figure 11.13.
Temperatures necessary to kill pathogens and other plant pests. Most of the temperatures are for a 30-minute exposure under moist conditions.

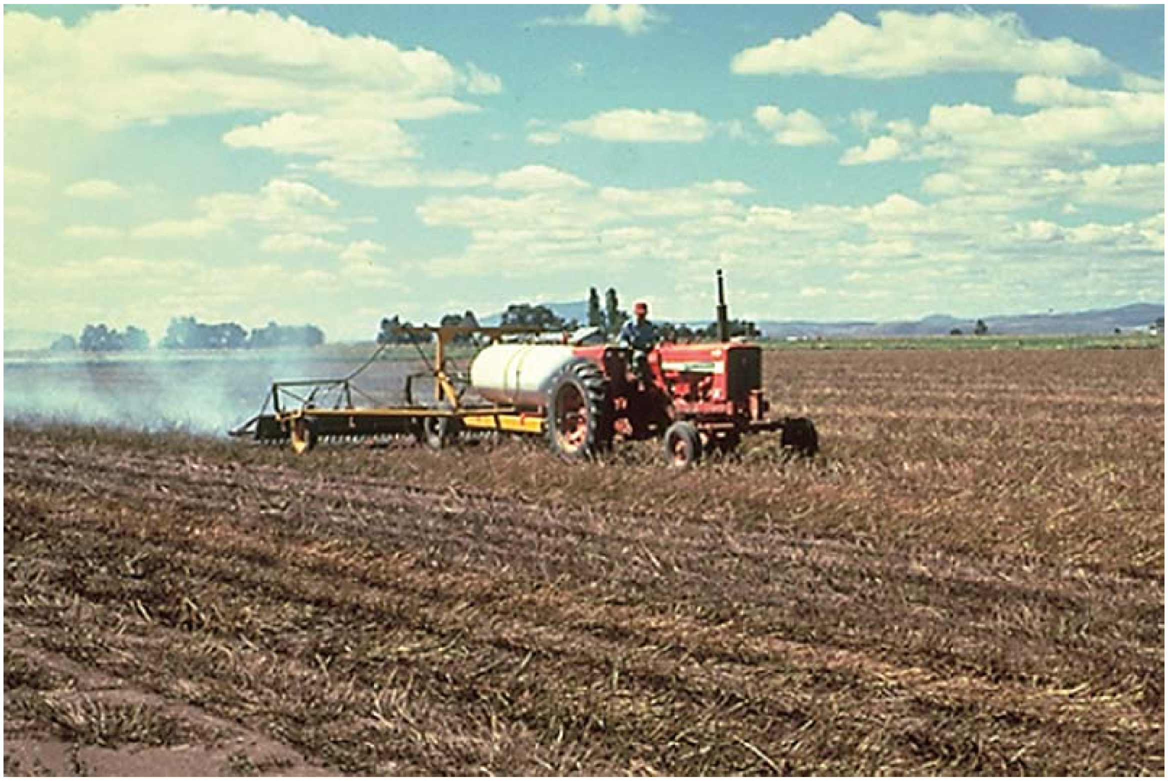


Figure 11.14.
Propane flaming of peppermint debris after harvest reduces *Verticillium* inoculum entering the soil.

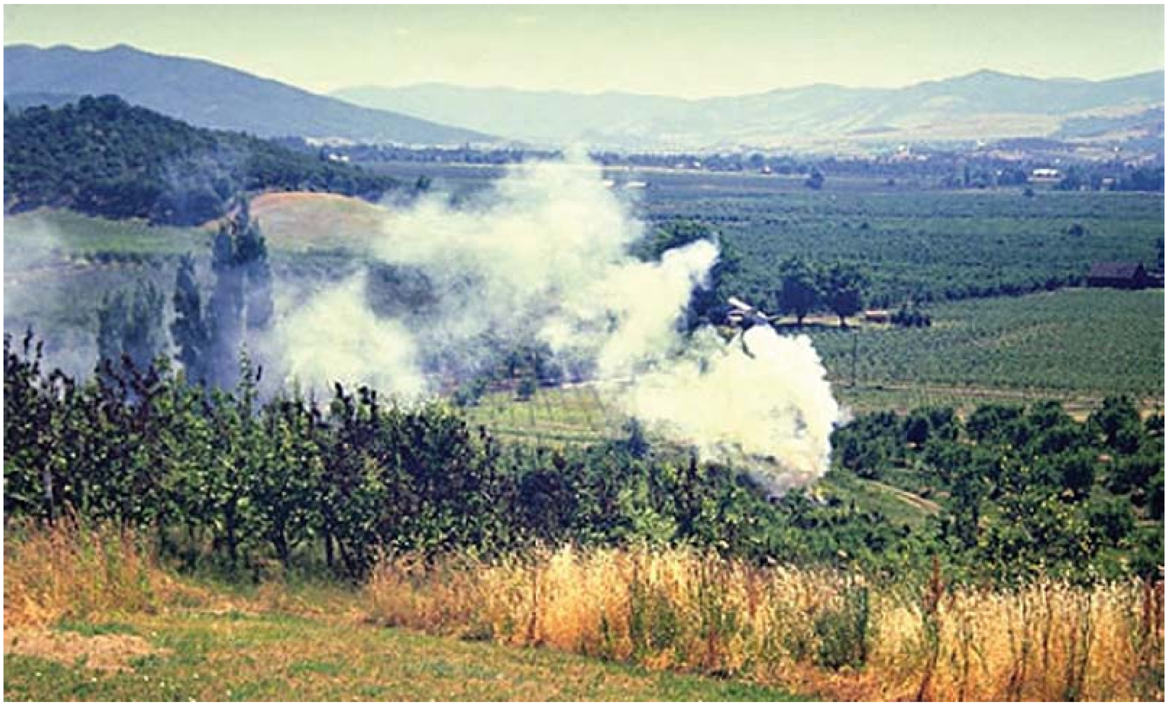


Figure 11.15.
Burning of fire blight prunings in a pear orchard.

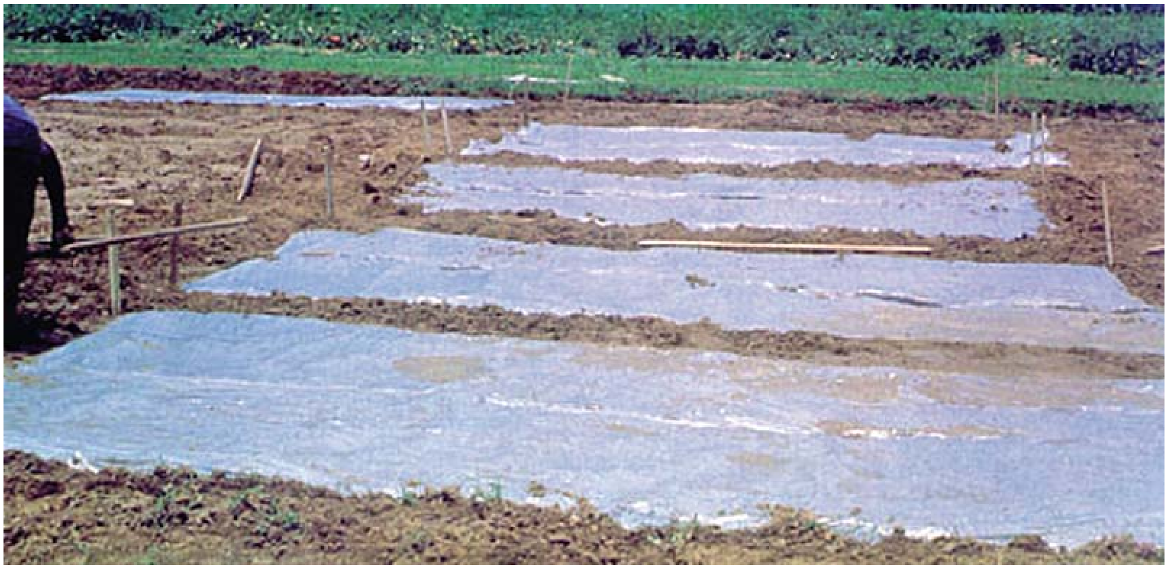


Figure 11.16.
Soil solarization. Soil is treated by covering it with clear plastic sheeting for 4-8 weeks.

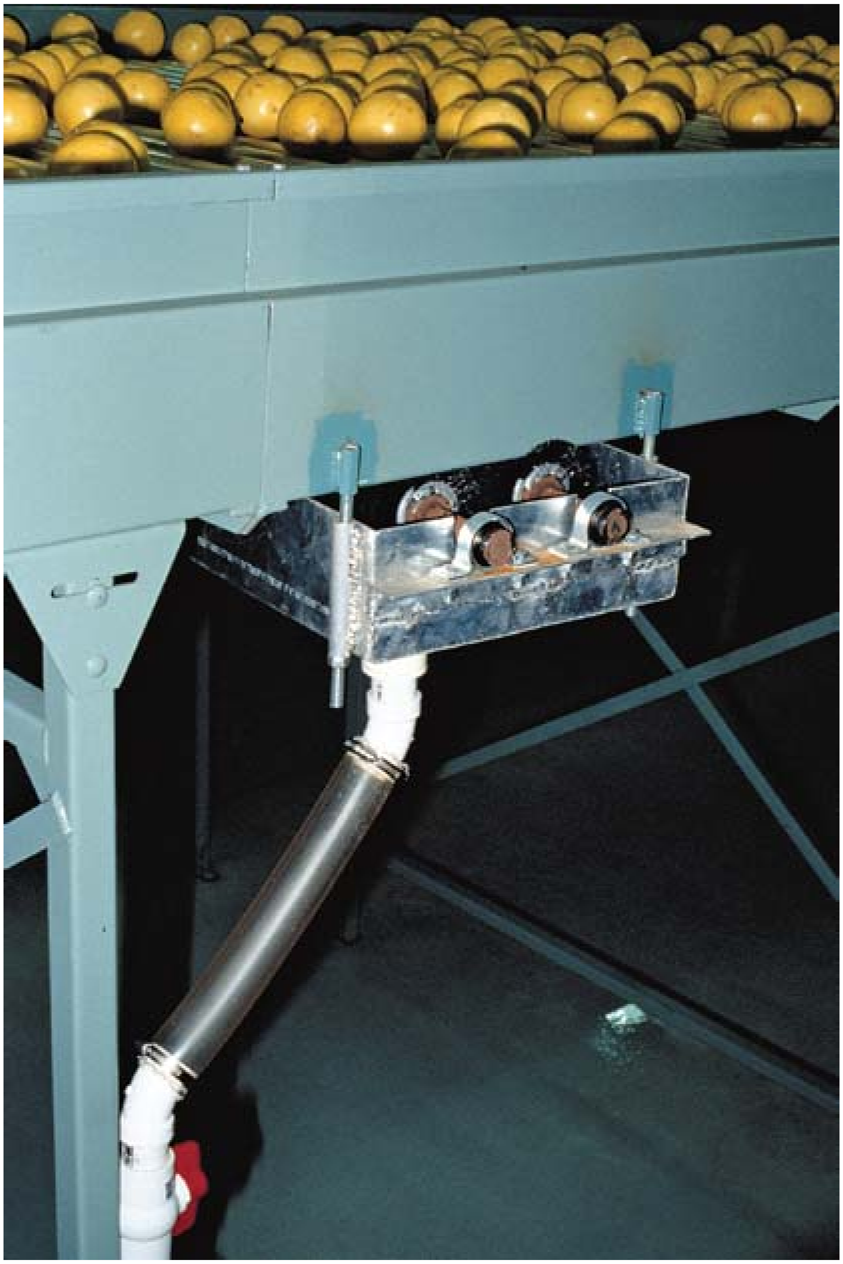


Figure 11.17.
A cleaning unit with which a disinfestant is applied to fruit conveyor rollers for daily sanitation of the citrus packing line.

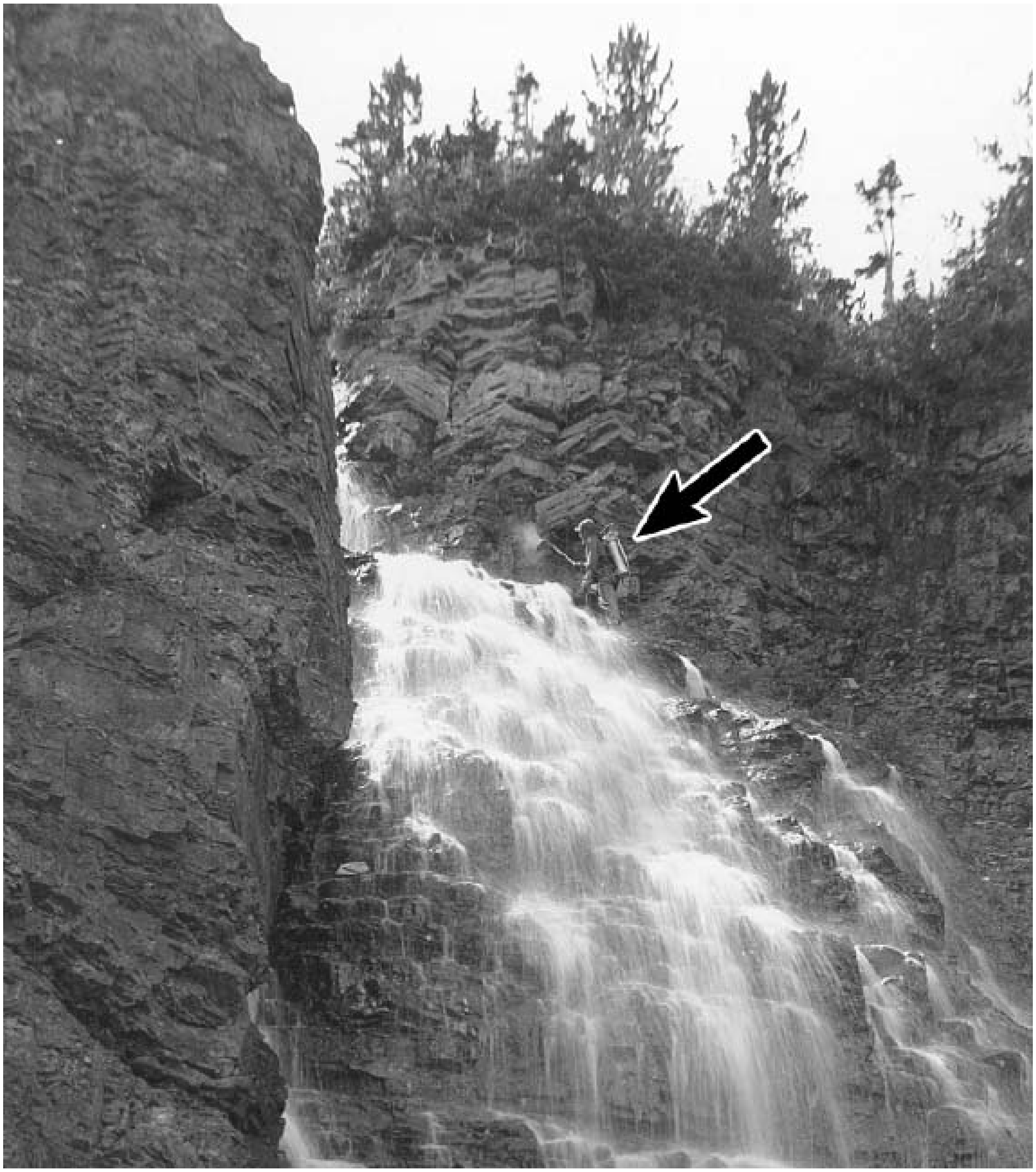


Fig. 11.18.
Herbicide application to a *Ribes* bush, the alternate host of the white pine blister rust fungus, along a waterfall in Glacier National Park. The applicator is shown by the arrow.

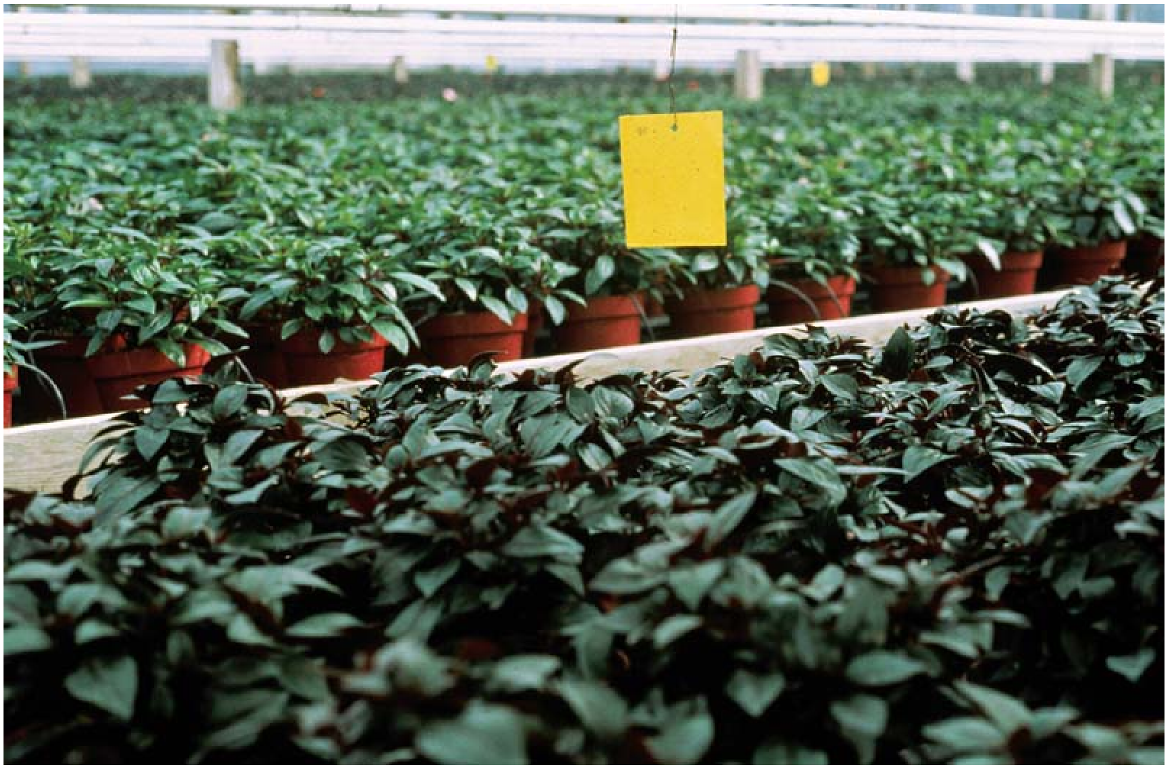


Figure 11.19.
Yellow sticky cards may be used to monitor greenhouse crops for virus vectors such as thrips and aphids.

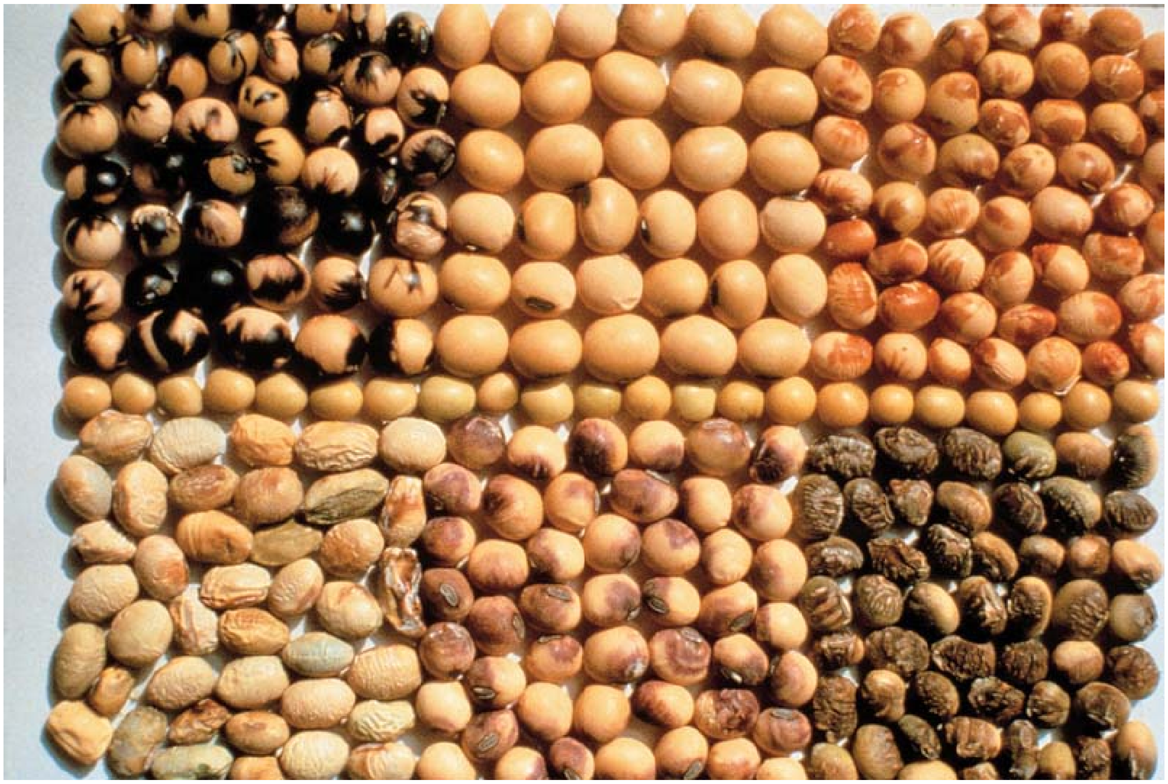


Figure 11.20.
Soybean seeds from plants infected by a virus (upper left and right) or by several different fungi (lower rows). Seeds in the upper center area appear healthy.

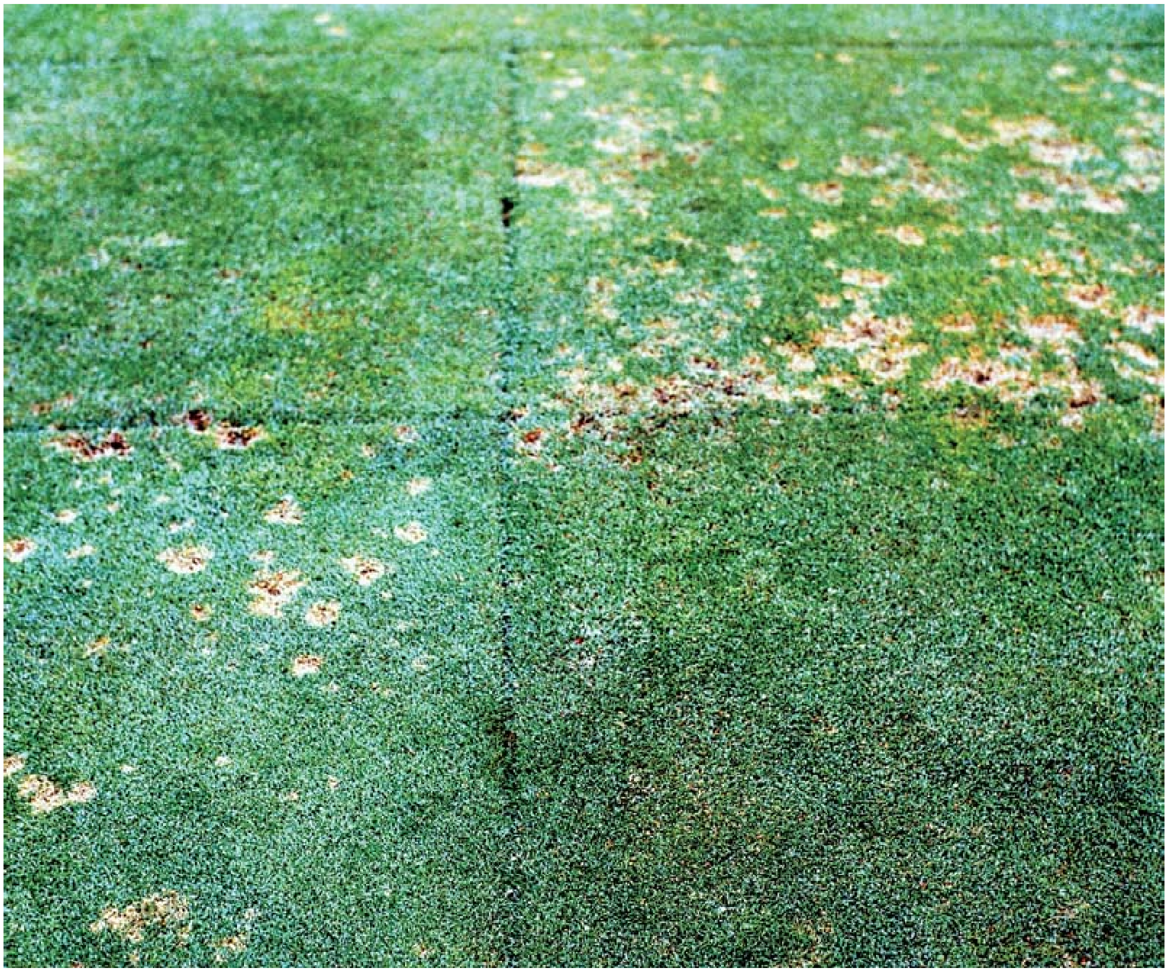


Figure 11.21.
Reduction of dollar spot on turfgrass in plots to which compost has been applied (upper left and lower right).

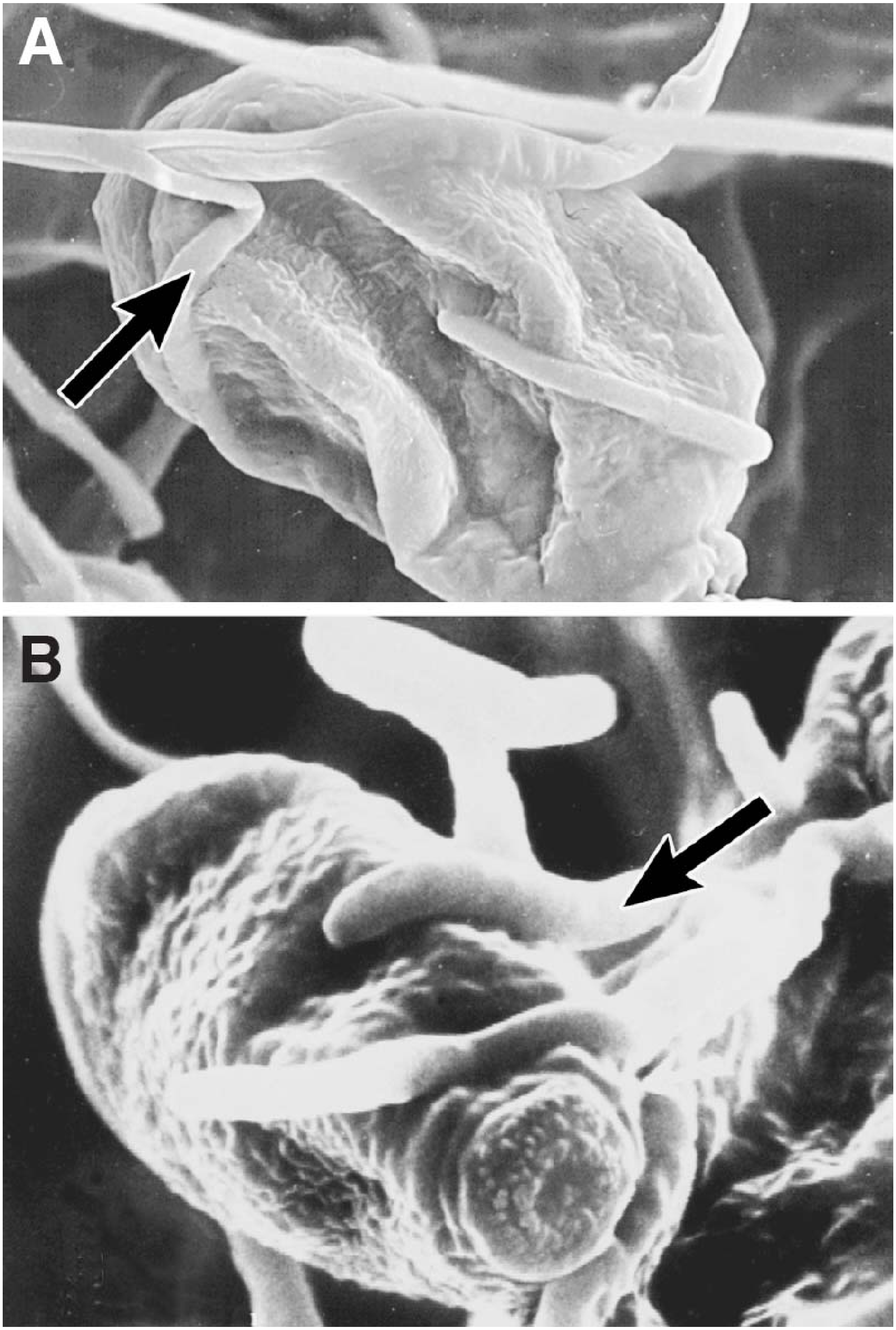

A
B

Figure 11.22.
Fungi that offer biological control of powdery mildews using two different modes of action to attack the mycelium and conidia of the pathogen. **A**, *Verticillium lecanii* is a hyperparasite. Note that the hyphae (arrow) penetrate and parasitize the conidium. **B**, *Pseudozyma flocculosa* (arrow) is an antagonist that causes collapse of fungal structures but never penetrates the powdery mildew fungus.

Figure 11.23.
A chestnut tree in Michigan recovering from chestnut blight because of naturally occurring hypovirulent strains of the pathogen.

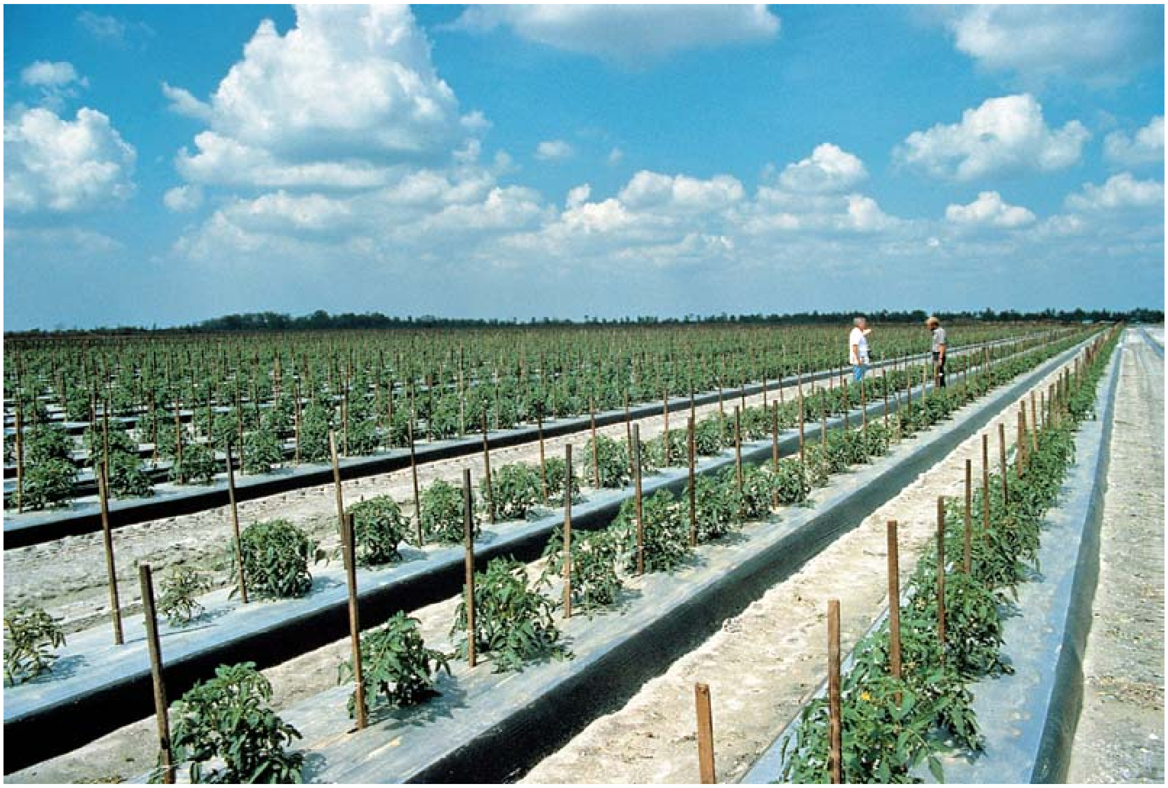


Figure 11.24.
Tomatoes staked and growing on plastic mulch to aid drying of plant surfaces and to protect plants from soilborne pathogens that might splash up from the soil.

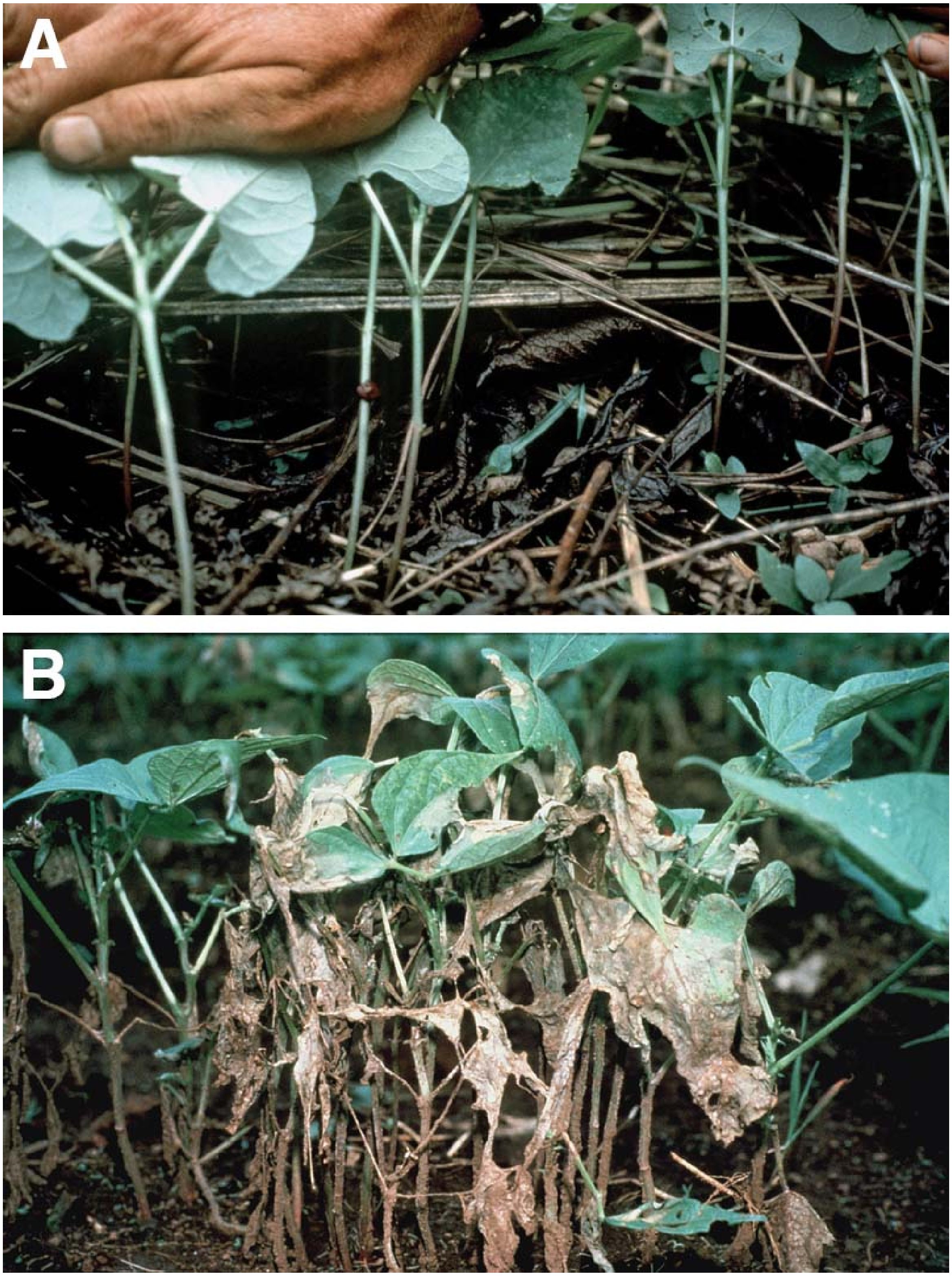


Figure 11.25.

Protection by a barrier. **A**, Use of organic mulch (a method called *frijol tapado*, “covered beans”) as a barrier to the soilborne fungus that causes web blight of beans (*Thanatephorus cucumeris*). The mulch also reduces weed growth and conserves soil moisture. **B**, Web blight of beans where organic mulch was not used.

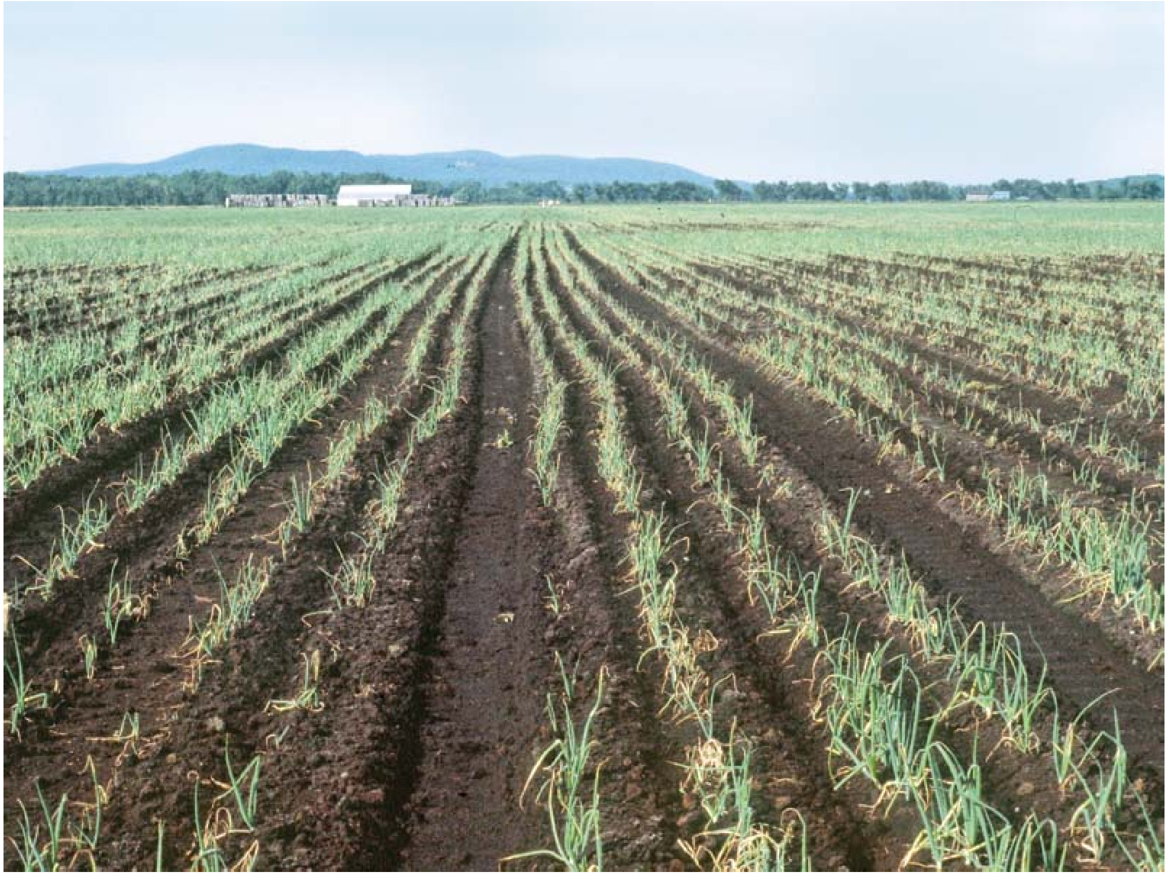


Figure 11.26.
Damage to onions caused by *Pythium* spp. in a field with poor drainage.

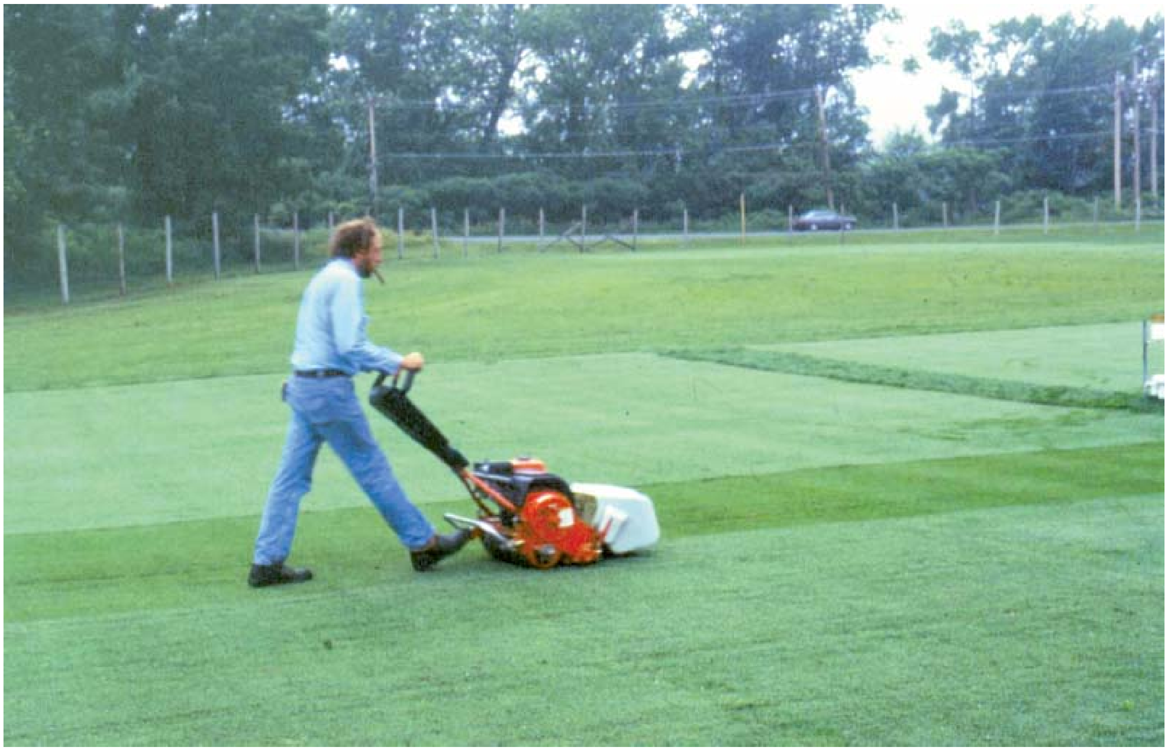


Figure 11.27.
Removal of dew on turfgrass by early morning mowing.

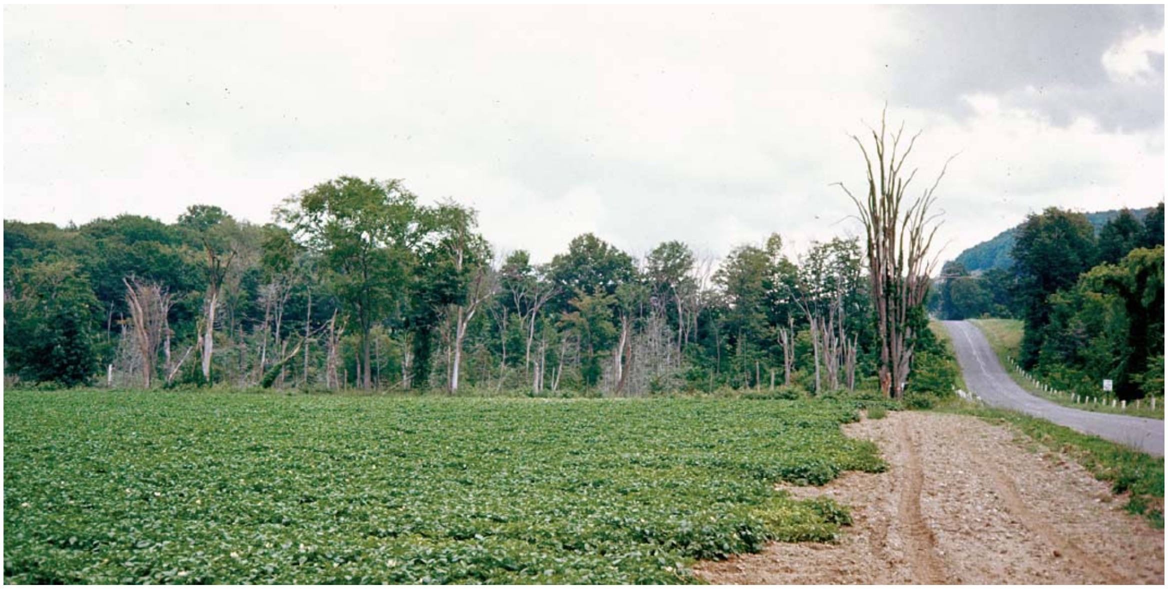


Figure 11.28.
Trees bordering this potato field may impede air movement and slow the drying of plants.

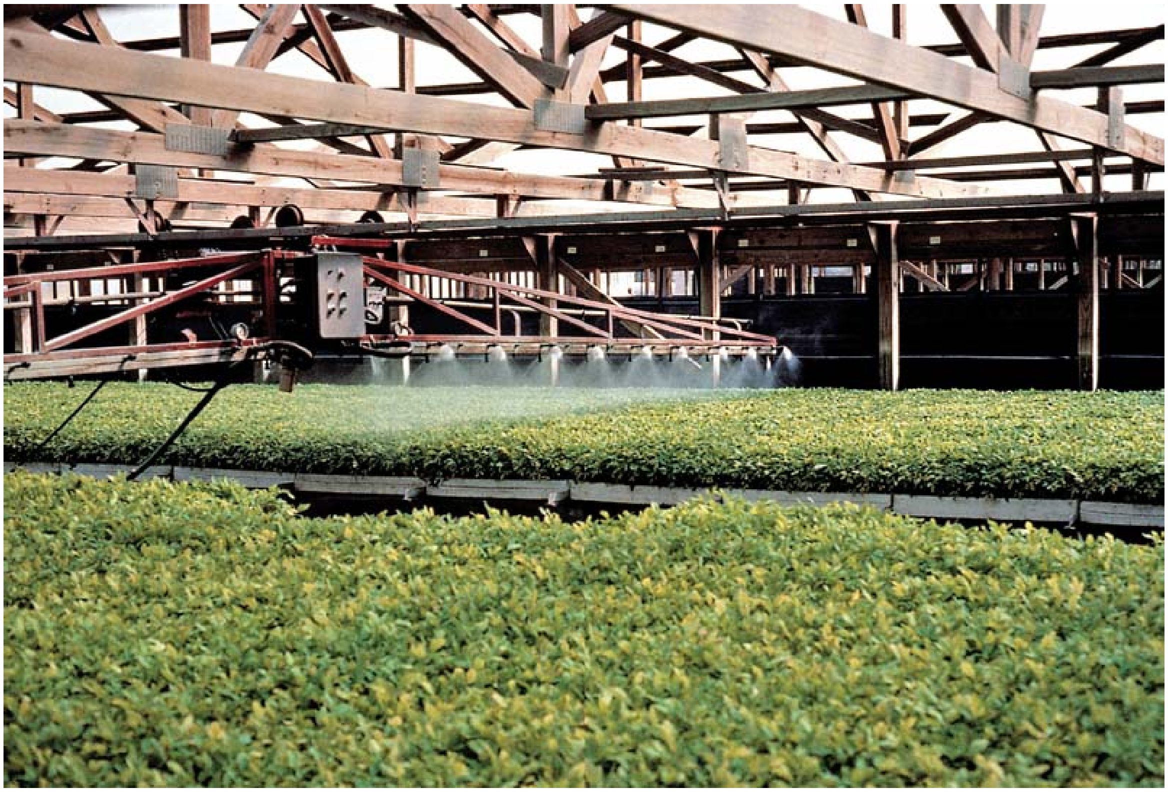


Figure 11.29.

Overhead irrigation and fertilization (fertigation) of greenhouse-produced tomato transplants. This can potentially contribute to pathogen dispersal and leaf wetness.

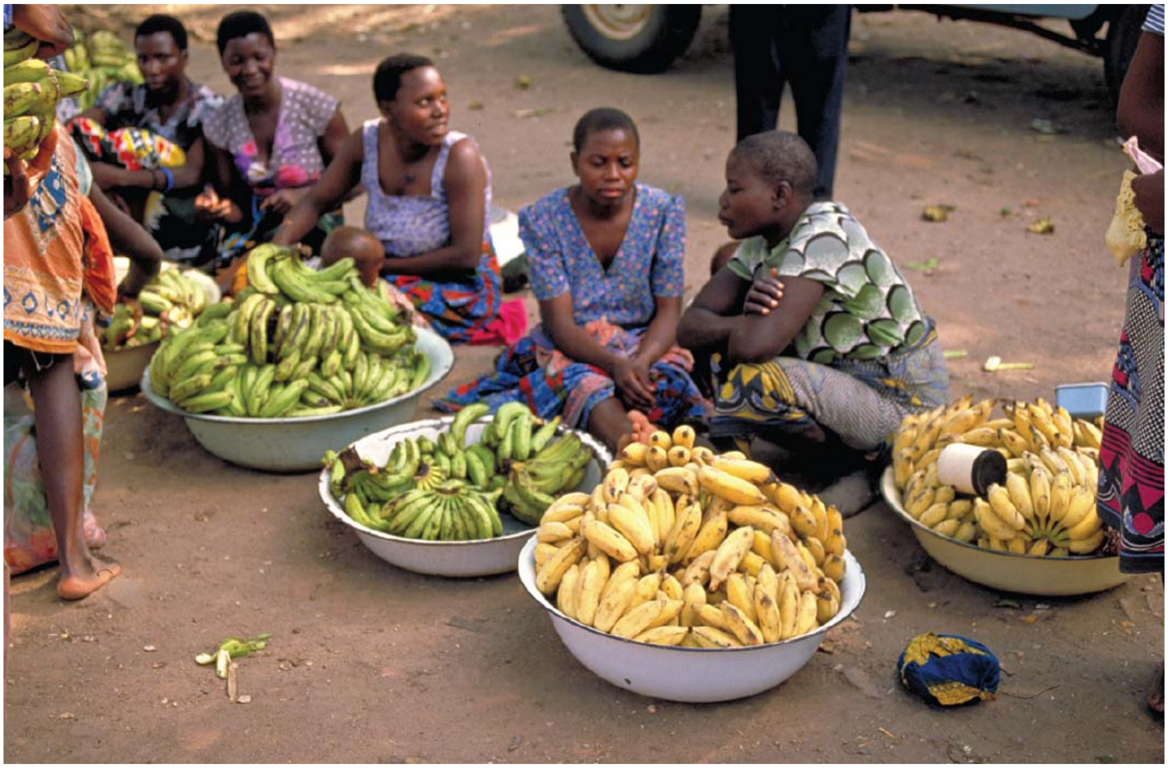


Figure 11.30.
Market in Malawi where several banana cultivars are for sale by traditional farmers.

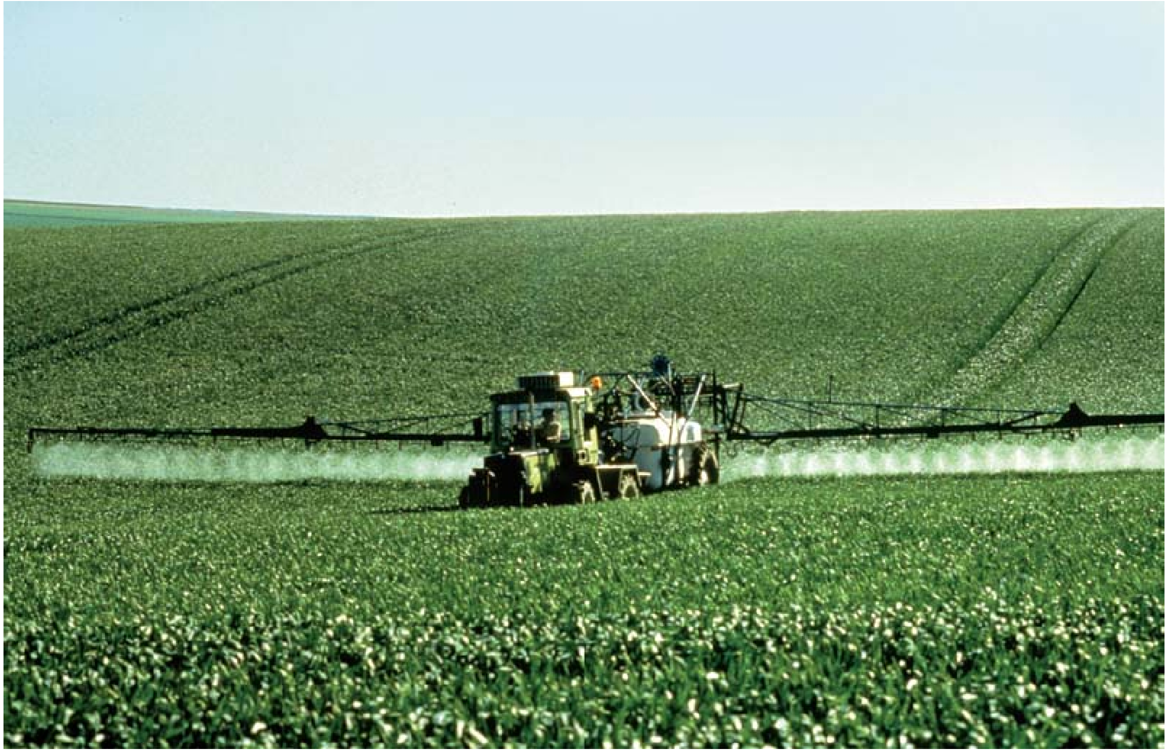


Figure 11.31.
Application of fungicide to a potato field.

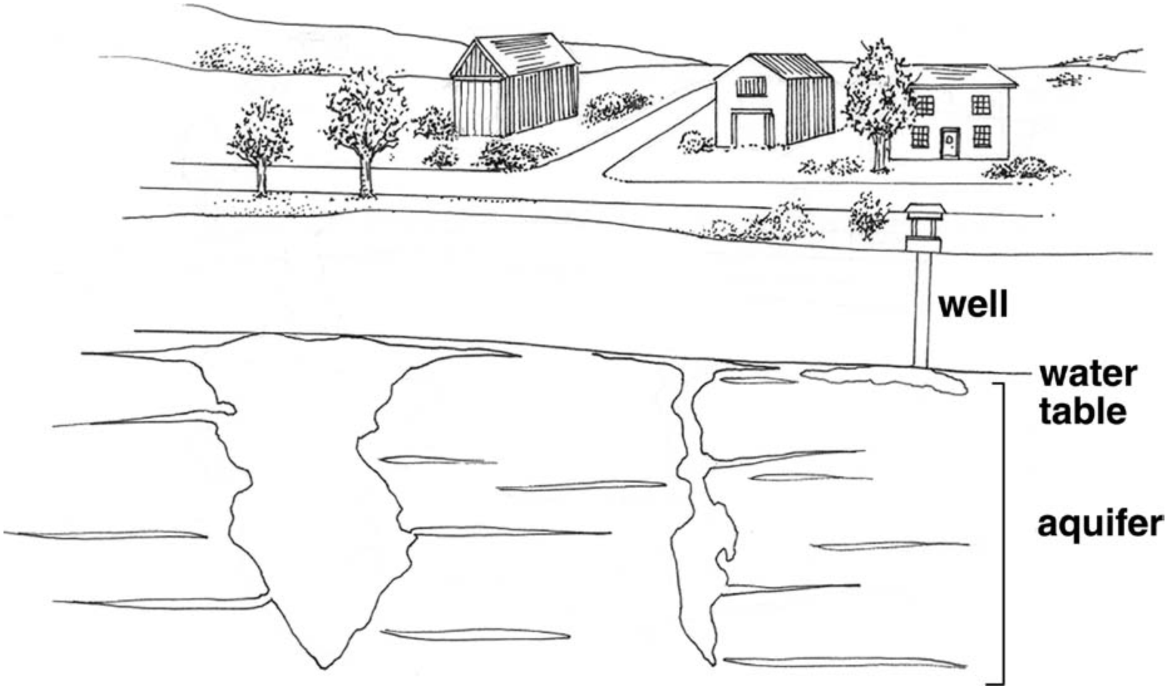

well
water table
aquifer

Figure 11.32.
Diagrammatic view of groundwater sources in aquifers.

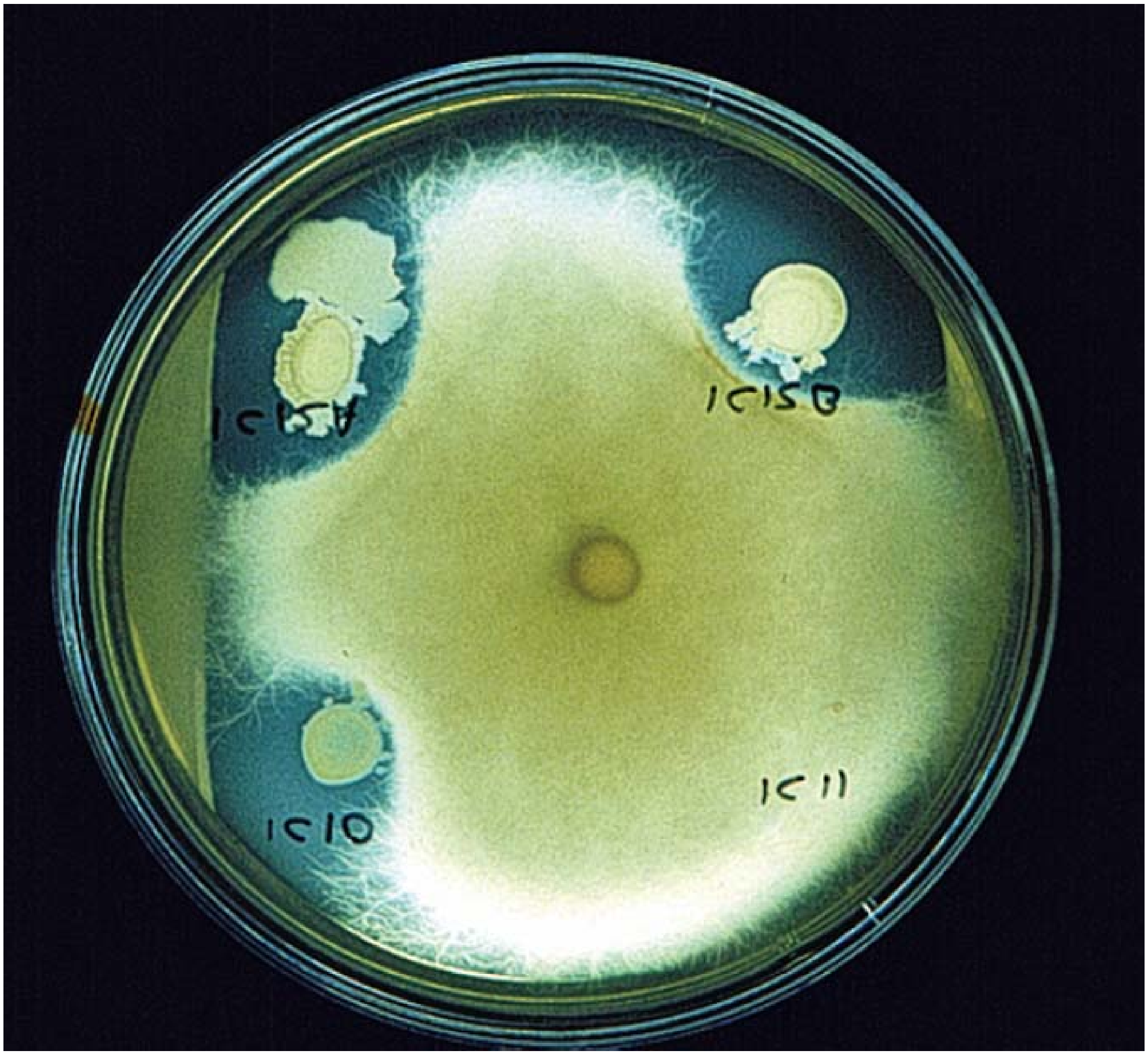

1C15A
1C15B
1C10
1C11

Figure 11.33.
Inhibition of *Gaeumannomyces graminis*, the fungus causing take-all root disease, by three different bacteria. The bacteria produce antibiotics that inhibit the growth of the fungus.

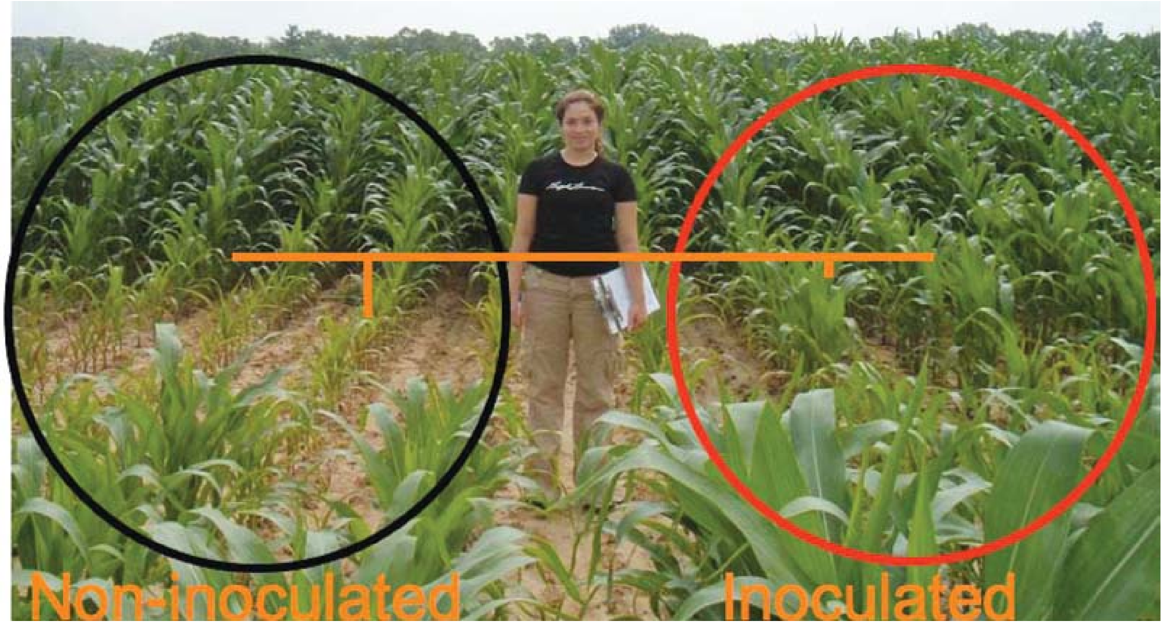

Non-inoculated
Inoculated

Figure 11.34.
Use of seed inoculation with rhizosphere-competent strains of *Pseudomonas fluorescens* under acidic soil conditions to improve corn growth in the field.

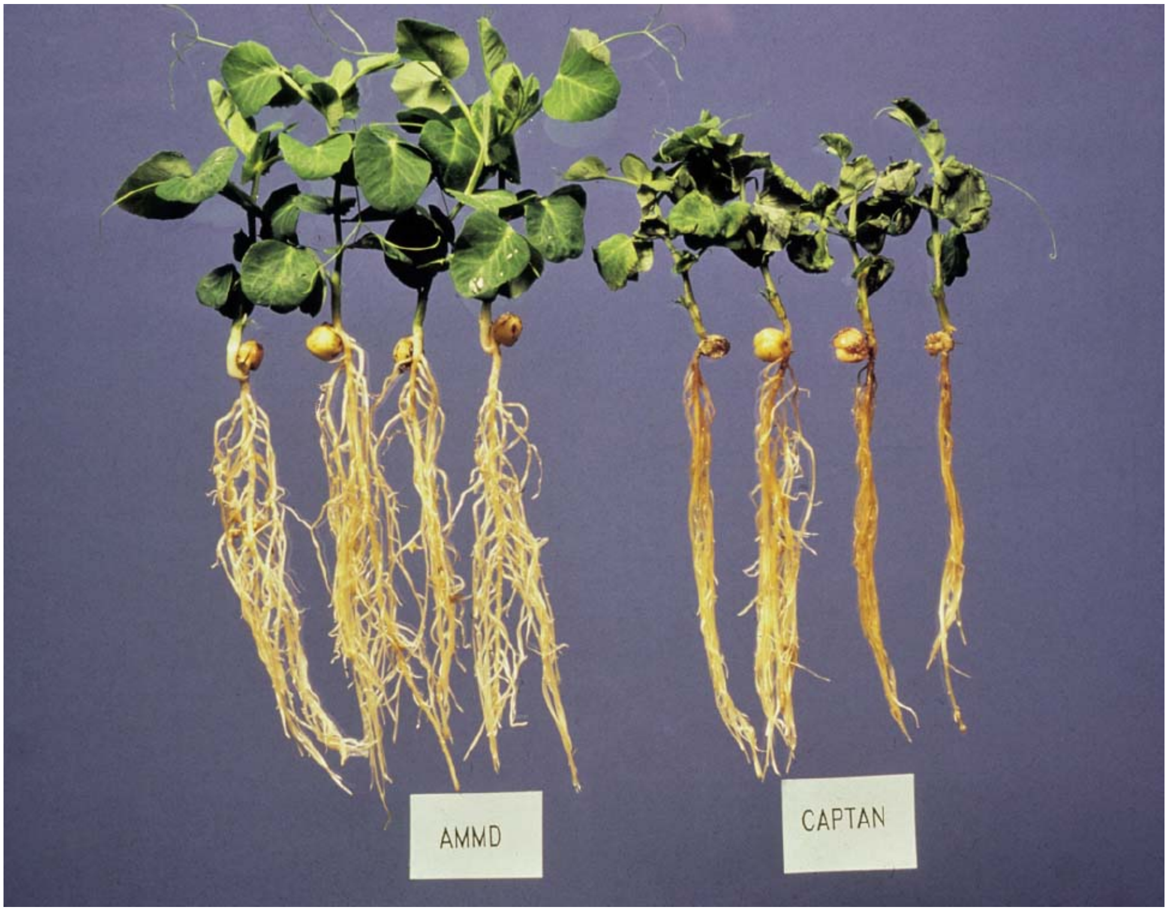


Figure 11.35.
Biological control of Aphanomyces root rot of pea. Left, pea seeds coated with the bacterium *Burkholderia cepacia* (*Pseudomonas cepacia*) strain AMMD. Right, pea seeds treated with the fungicide captan. All plants were inoculated with zoospores of the pathogen.

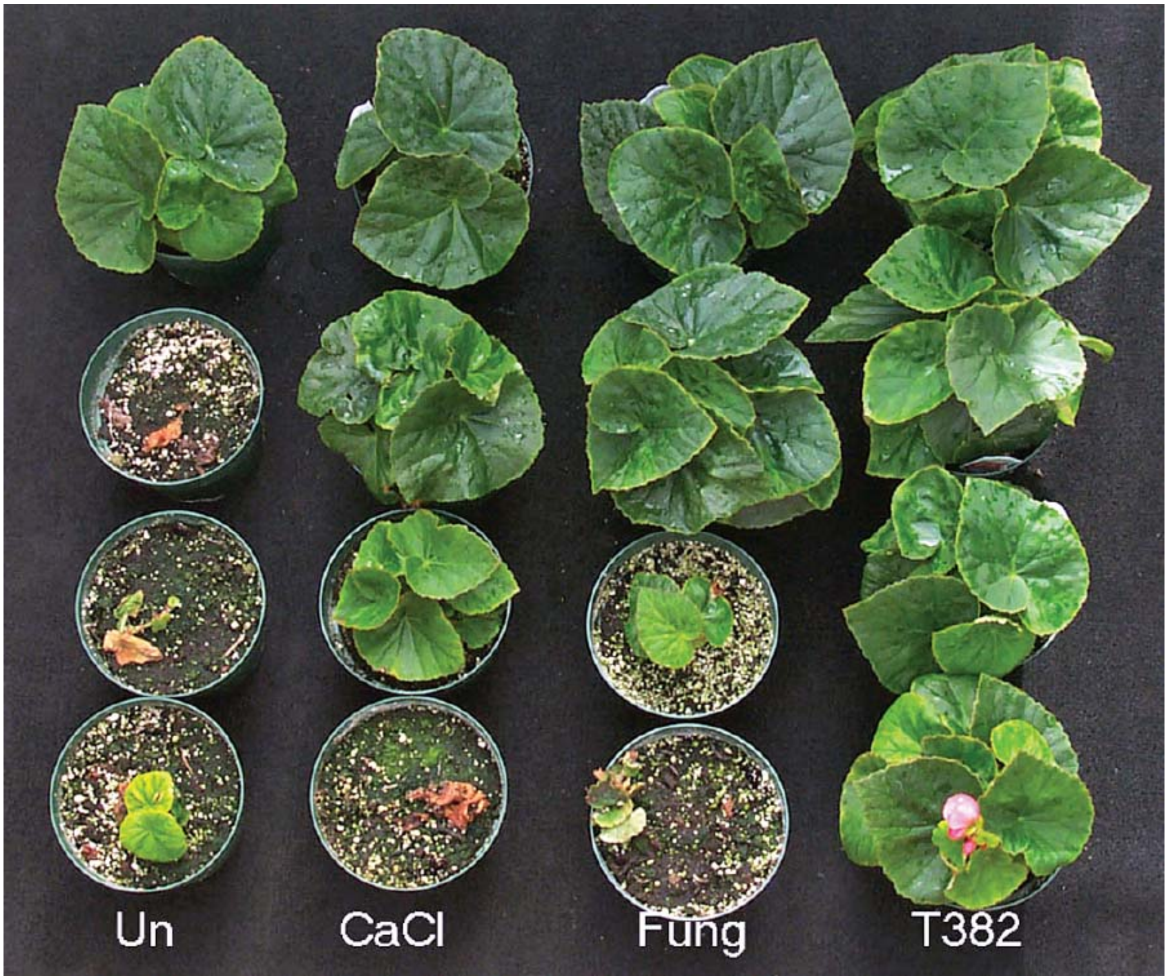


Figure 11.36.
Begonias grown in the greenhouse and inoculated with *Botrytis cinerea*, the fungus causing gray mold, under conditions optimal for the development of disease. Treatments (with four replications) from left to right: untreated (Un), $CaCl_2$, chlorothalonil fungicide (Fung), and a biological control fungus *Trichoderma hamatum* T382 inoculated into the potting mix (T382).

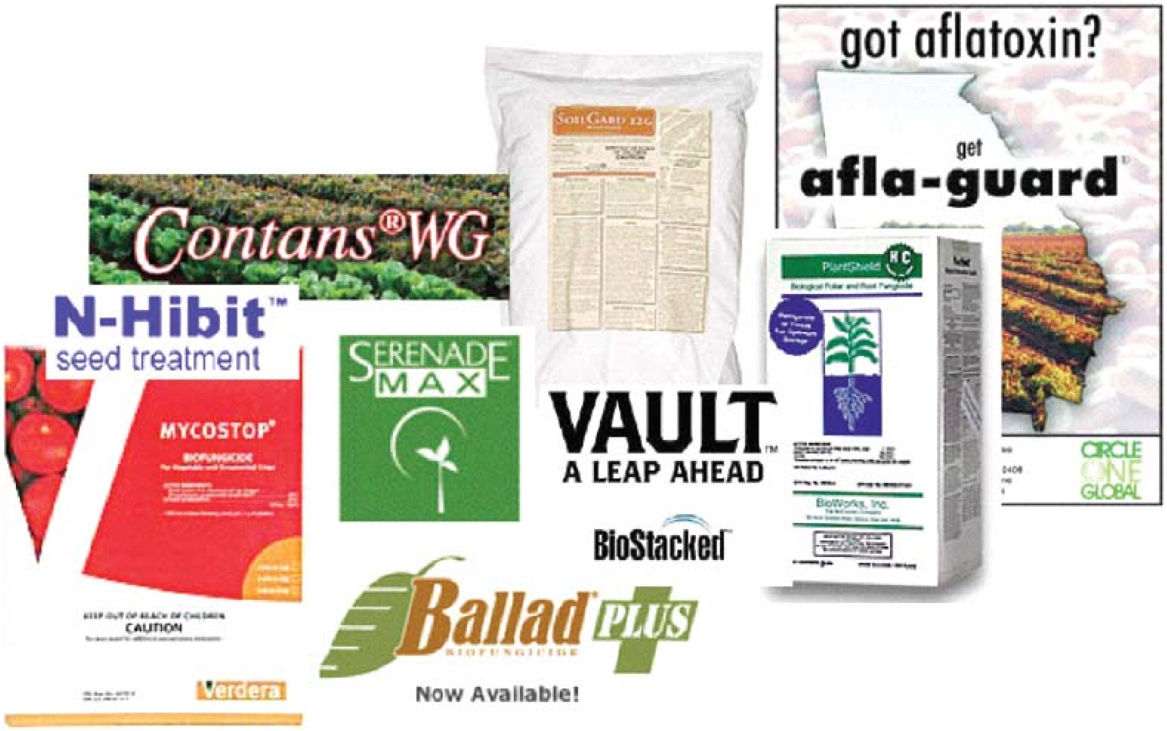

got aflatoxin?
get
afla-guard
Contans®WG
N-Hibit™
seed treatment
MYCOSTOP
BIOFUNGICIDE
KEEP OUT OF REACH OF CHILDREN
CAUTION
Verdera
SERENADE
MAX
SoilGard 12G
VAULT™
A LEAP AHEAD
BioStacked
PlantShield HC
BioWorks, Inc.
CIRCLE ONE GLOBAL
Ballad PLUS
BIOFUNGICIDE
Now Available!

Figure 11.37.
Some examples of commercial biological control products (biopesticides).

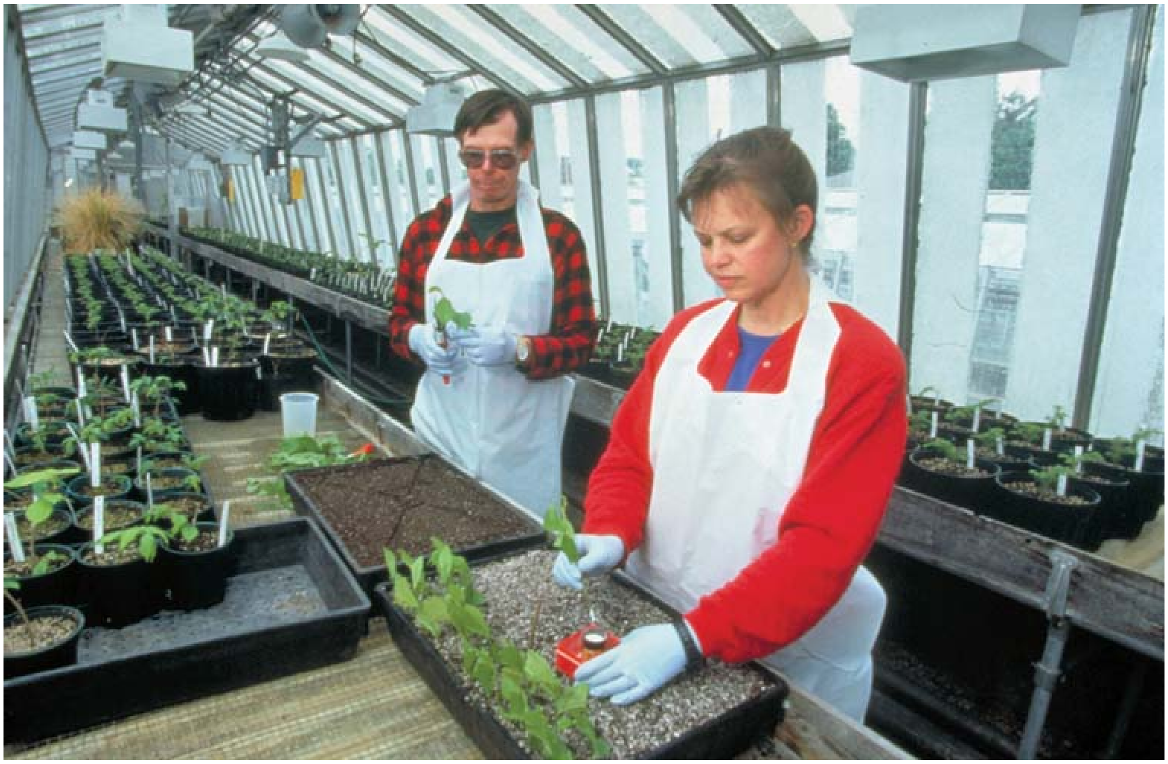


Figure 11.38.
Elm seedlings in the greenhouse prior to testing for resistance to Dutch elm disease.

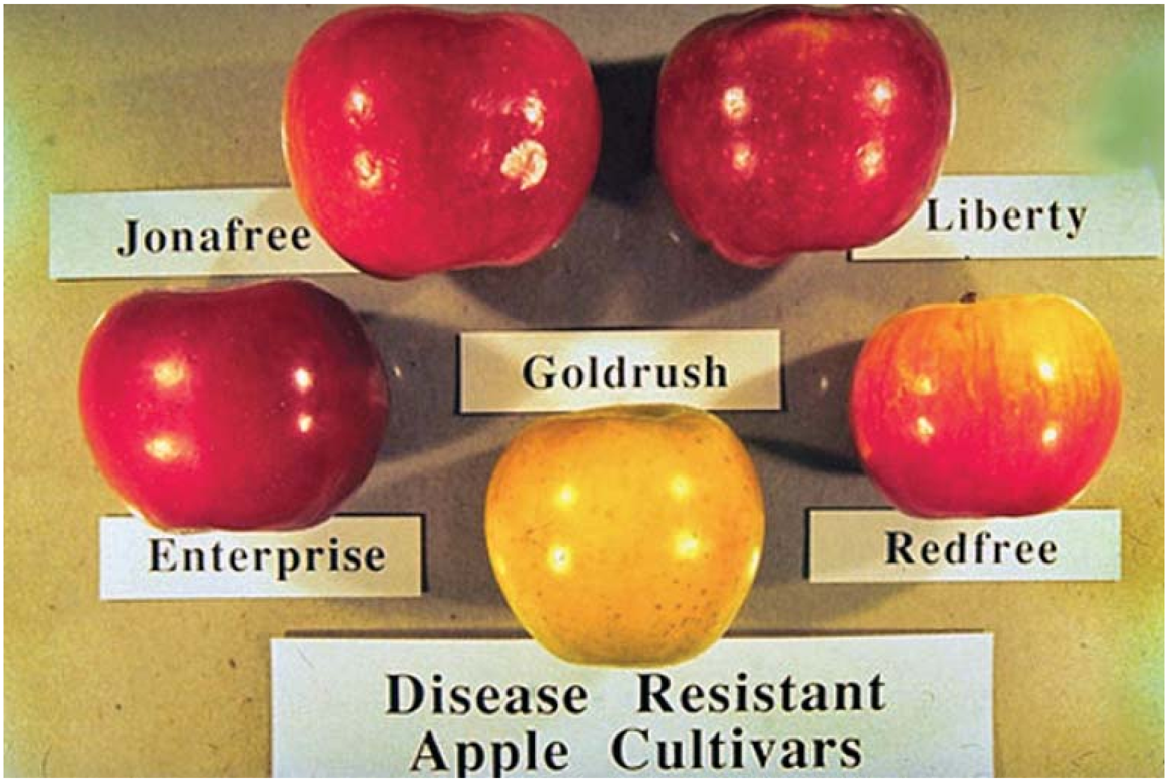

Jonafree
Liberty
Goldrush
Enterprise
Redfree
Disease Resistant
Apple Cultivars

Figure 11.39.
Some apple cultivars that are resistant to apple scab.

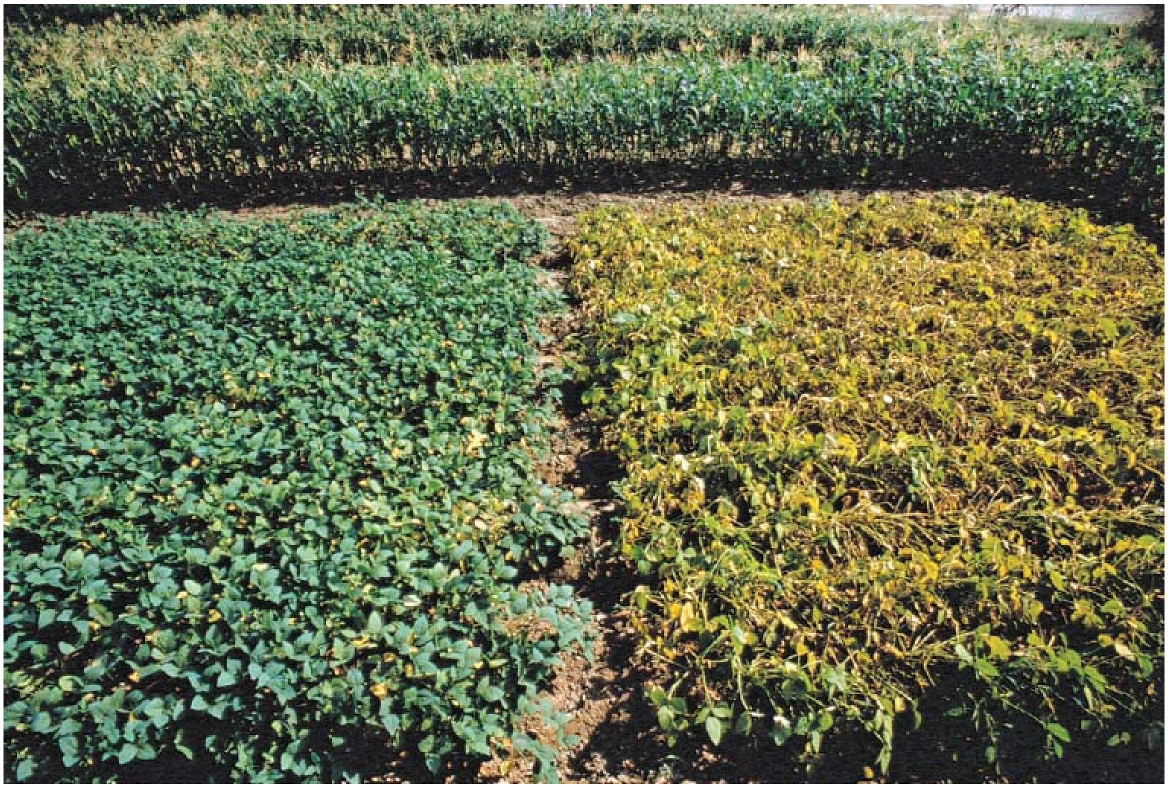


Figure 11.40.

Experimental plots with plants tolerant (left) and susceptible (right) to soybean rust.

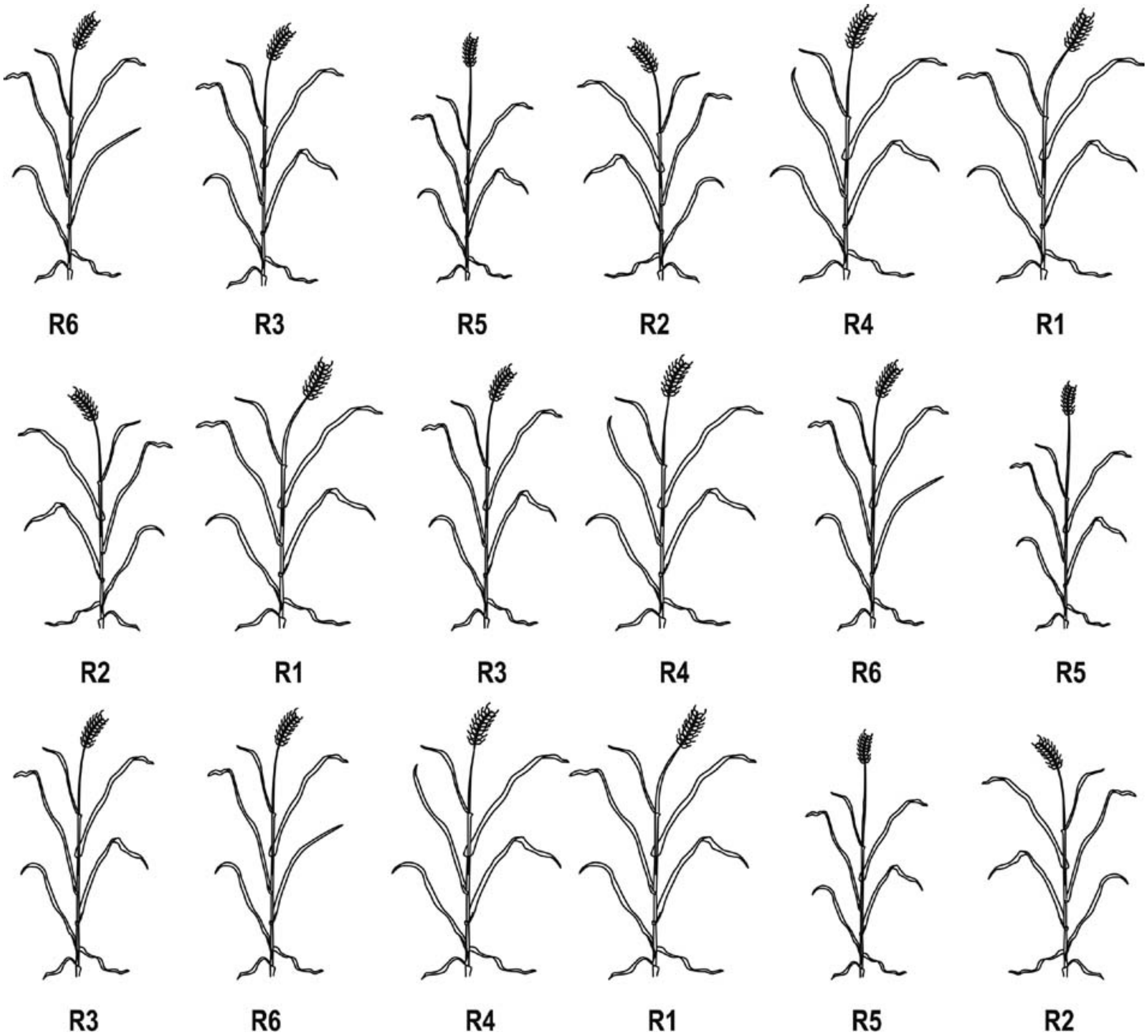

R6
R3
R5
R2
R4
R1
R2
R1
R3
R4
R6
R5
R3
R6
R4
R1
R5
R2

Figure 11.41.
Diagram of multilines of a cereal crop showing a mixture of six R genes planted out randomly.

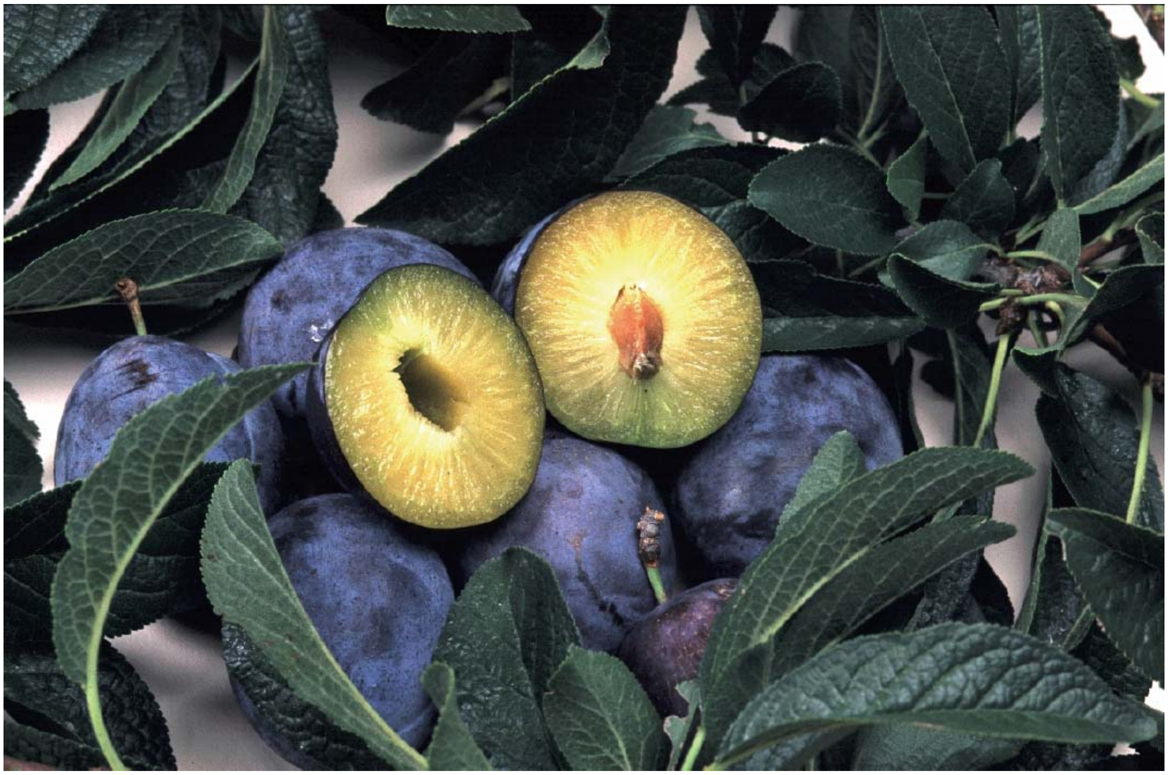


Figure 11.42.
Resistance to *Plum pox virus* in transgenic plum clone C5 due to the insertion of the PPV coat protein gene into the plant genome.

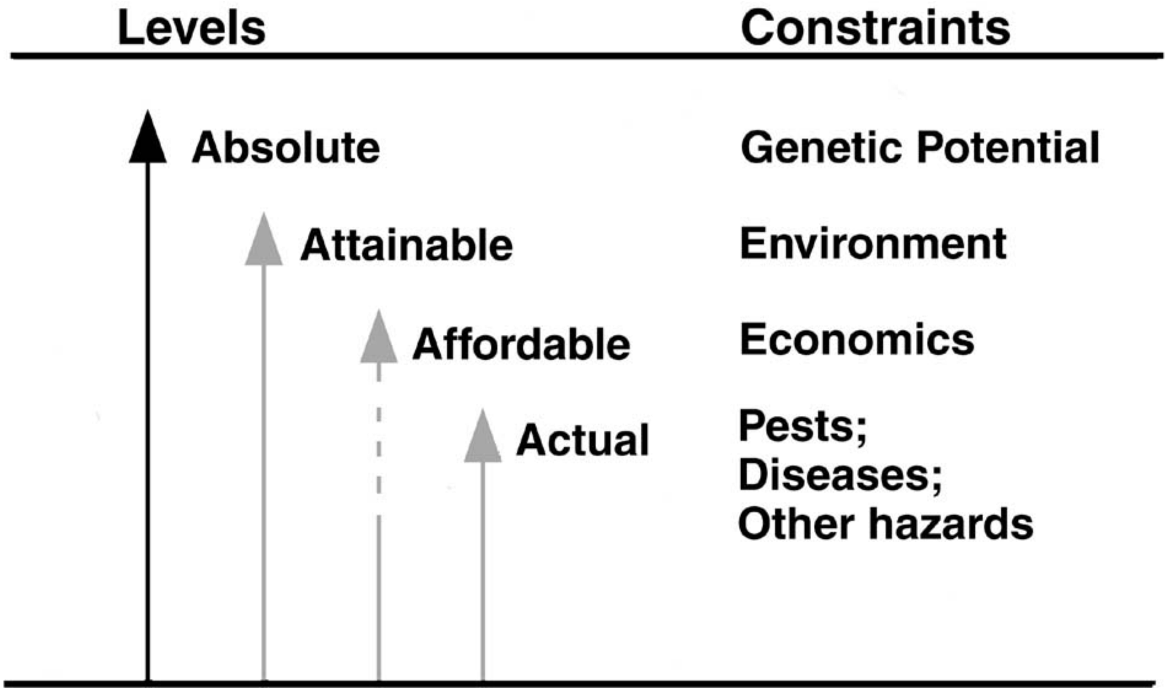

Levels
Constraints
Absolute
Genetic Potential
Attainable
Environment
Affordable
Economics
Actual
Pests;
Diseases;
Other hazards

Figure 11.43.
Constraints to crop production. See discussion in text.

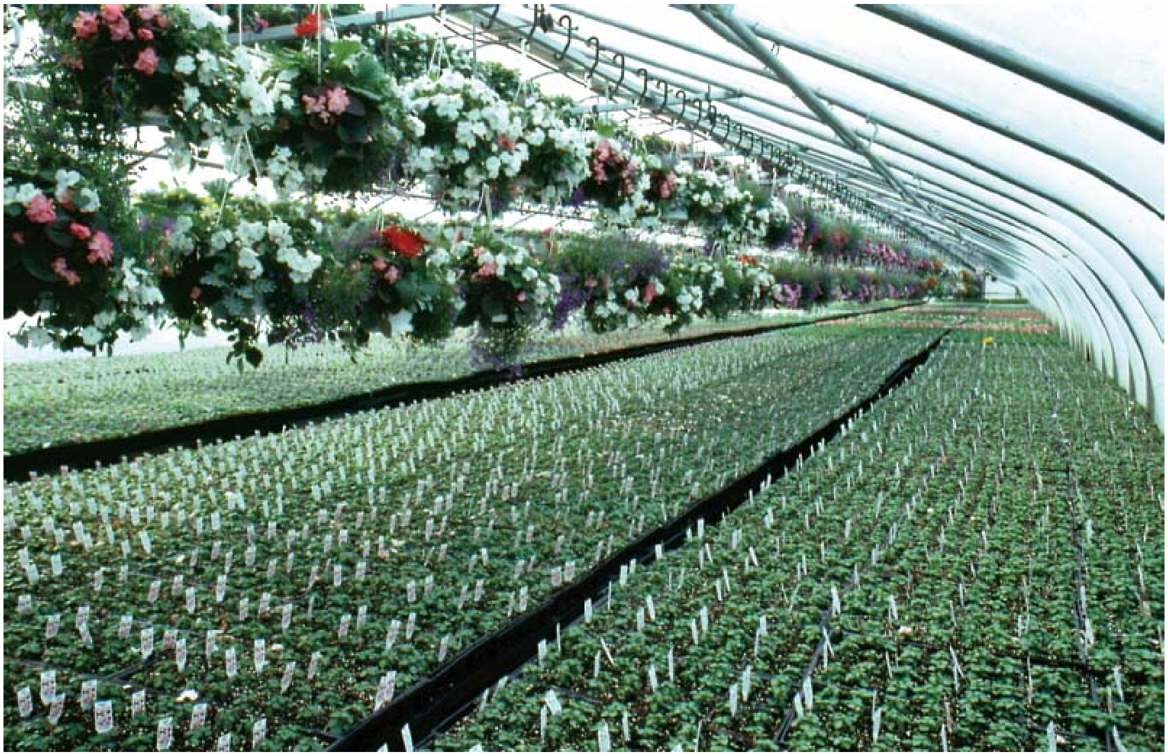


Figure 11.44.
Modern agriculture puts plants at risk with genetic uniformity, as illustrated by this greenhouse full of genetically uniform geraniums.

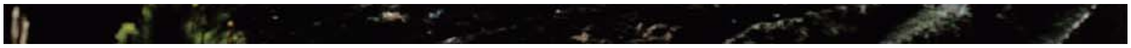


Figure 11.45.
Rice culture in the highlands of the Philippines. Such intensive agriculture can increase the risk of plant diseases.

A
B

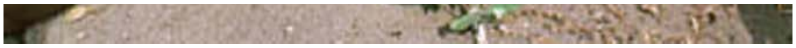


Fig. 11.46.

A, Harvest of cassava (manioc) (*Manihot esculenta*) roots, an important crop in the tropics but not commonly consumed in temperate areas. **B**, African children harvesting cassava leaves. Crop diversity is important in providing a stable world food supply.